Library

Vol. XX., No. 7.]

JUL 13 1927

[May, 1927.

PROCEEDINGS

OF THE

ROYAL SOCIETY OF MEDICINE

LIBRARY

LONGMANS, GREEN, AND CO. LTD.
39 PATERNOSTER ROW, LONDON
NEW YORK, BOMBAY, CALCUTTA, MADRAS

All rights reserved

ELECTRO-COLLOIDAL GOLD & SILVER.

ORARGOL (A.F.D.)

AMPOULES 5 c.c. and 10 c.c.

ORARGOL is capable, injected in time and in suitable dosage, to cut short a serious case of pneumonia, influenza, or erysipelas. In all febrile conditions, whatever the cause, it may be used without hesitation, being absolutely inoffensive and free from toxicity. Infections in which ORARGOL injections have been successfully employed are: Influenza, bronchitis, pneumonia and broncho-pneumonia, endocarditis and infectious endocarditis, acute articular rheumatism, acute infectious hepatitis, epidemic encephalitis, puerperal affections, kidney abscess.

ENCEPHALITIS LETHARGICA

AN INTERESTING REPORT:—

"A case of Encephalitis Lethargica of severe type, treated successfully by injections of Electro-colloidal Gold and Silver."

"The Lancet," July 24th, 1926, p. 172.

ORARGOL
COLLOIDAL
GOLD & SILVER

FORMS AND INDICATIONS:

ORARGOL AMPOULES, 5 c.c. & 10 c.c.	Acute and Chronic Infectious Conditions (Septicemia, Pneumonia, Influenza, etc.)
ORARGOL (in bulk).	Internal and External Antisepsis (Intestinal Intoxication, Wounds, etc.)
ORARGOL OUTFIT	Oto-Rhino-Laryngology.

Sample of Orargol Ampoules and full particulars from—

THE ANGLO-FRENCH DRUG CO., LTD.,
238A, GRAY'S INN ROAD, LONDON, W.C. 1.

Section of Balneology and Climatology.

President—Dr. R. Ackerley.

DISCUSSION ON THE BREAKDOWNS OF MIDDLE LIFE.

Dr. J. Campbell McClure.

In our mechanical age premature breakdowns will be more easily comprehended by ordinary people than formerly, when opportunities for the study of such happenings were small. Every one, for instance, knows that the breakdown of a motor-car is due to four causes: faults in construction, bad material, undue stress without sufficient rest, and mishandling of the machine.

It is the same with the human body. There are those who maintain, with some justice, that man has not yet adapted himself satisfactorily to the upright position; that even to-day the more unnatural forms of food still induce anaphylactic phenomena extremely unpleasant to the consumer; and that there are those who by their reactions take us back to a time when all ingested material was a poison which either killed or gave an immunity that afforded a new chance of advancement to the survivor. Those inherent faults in our construction are still with us after millions of years since man first appeared on the earth, and are being slowly eliminated and corrected, less by the ephemeral efforts of enthusiasts than by the slow response of tissues to changing conditions. Only in one experience in human life is this aspect important to-day, the successful resistance of our tissues to specific infections, especially in epidemic form. Our faulty construction is apparent in that our tissues, in most instances, are not naturally capable of dealing successfully with massive infections, but have to be trained before that capacity is effectively developed. This is a point that many enthusiasts ought to remember before insisting on the sun-lit, microbe-free, dietetically perfect life, in spite of which certain communities in recent times have been almost wiped out by simple epidemics such as measles.

Bad material in the construction of the human body is, perhaps, scarcely so deadly as in a machine, because in the case of living tissues, faulty material can be improved and often converted into first-grade stuff if the fault is discovered early. Even when the fault is discovered early in middle life, the faulty tissues may be so nursed as to ensure the efficient functioning of the human mechanism for the full term of a working life.

Undue stress without sufficient rest is a very fruitful source of premature breakdown. The saying that "work never kills" is simply silly. Work may and will kill if the body is not rested so as to allow sufficient time to permit elimination to get square with katabolism. True breakdowns from overwork alone are not common, but breakdowns from work in conjunction with faulty constructional material are very common, and when, by the occurrence of such a breakdown, the physician discovers the fault, the breakdown may be rightly considered later on to have been a very beneficent episode in a worker's life.

The most irritating breakdown from the physician's point of view is that which occurs in a machine made of good material, not overworked, but mishandled by a

careless, stupid, or ignorant person. Ignorance is behind many breakdowns; but when this lack of proper information is associated with reasonable intelligence, the case is never hopeless. When, however, ignorance is combined with irresponsibility, the lesson of the breakdown is forgotten too soon to be effective in the prevention of a future collapse, at least until the effects of carelessness have so damaged tissues as to make repair impossible. The later stages in such cases are extremely sad, when to physical failure is added remorse for the failure to exercise control.

Of all breakdowns the most common are those due to the failure of tissues successfully to combat a stress apart from any gross carelessness or abuse. All observers will agree that certain people are from the beginning of their life more susceptible to prevalent infections than others, more liable to fatigue, either mental, or physical, or both, who in the early years are left behind by their more powerful and active competitors. Their level of activity is low. On the other hand, it is equally apparent that in a large number of cases the hustling young man tends to slow down very noticeably in early middle-age, and may even be incapable of new impressions in the early forties. Similarly we are all familiar with the sudden death or nervous breakdown of the apparently physically robust person in early middle life. In contrast to this is the steady maintenance or even increase in capability and powers of achievement in middle life of those who had difficulty in their earlier years in keeping up to average capacity and attainment. It would appear that the slow starters, lacking something that enables many of their associates to overtake a larger amount of work than they, are saved from overstraining themselves by a knowledge of their breaking point forced on them by early and repeated experience, so that they are able to nurse carefully their faulty material to a point far beyond a large number of their more striving and robust compeers. Thus they survive middle life with less risk of serious breakdown than many apparently stronger machines. Middle age is for most of us a time of failure of power in one way or another, and when by reason of happy experiences in the past a man goes on using a forty-five to fifty year old brain and body exactly as if it were thirty or thirty-five, a time comes when a perfectly good average mechanism will break or show signs of premature wear because the period of full activity, mental, bodily and emotional, is carried beyond the normal point of safety. The limitations of tissue capacity must be realized if a full term of working life is to be expected. After complete development, the older the tissues, the less able are they to endure long strain without rest, or to recover quickly from the stress of sudden and severe emergency. They can do good work, but they require more careful handling than younger tissues.

In different groups of the middle-aged, the same strain does not hurt the same tissues. Pyorrhœa, for example, may in one group determine arthritic changes, in another fibrositis, in a third cardio-vascular degeneration, in a fourth nervous disturbances, in a fifth disorder of the digestive mechanism, either gross or functional, and so on.

It seems to me that, in middle-age, breakdowns are due to two main causes: a local vulnerability of tissues which varies in different individuals, and a general failure in the vitality of the organism as a whole. The failure of vitality lays the organism open to attack; the more local vulnerability determines the incidence of the πληγή.

The normal failure of power in middle-age is, in my mind, largely due to the failure of certain endocrine secretions, notably those of the thyroid gland and the gonads. When, to this, normal failure is added the disturbance produced by the continuance into middle-age of the habits and diet of the later period of youth, which are often extraordinarily indiscreet, breakdowns more or less serious occur, affecting one or other somatic system according to the personal vulnerability of the patient.

When to the normal endocrine failures of middle-age are added other endocrine weaknesses, many of which are congenital, the difficulties of the physician are increased, and in the present state of our knowledge it is often hard to determine without therapeutic experiment what endocrine glands need reinforcement. Such empiricism is absolutely justifiable, indeed without it progress would be impossible. If this question of endocrine faults or balance were all that was to be considered in the breakdowns of middle-age, the subject would be much simpler. There remains the question of that vulnerability of tissues which determines the incidence of the stroke that gives our picture of a disease.

The evidence before us at present does not warrant the assumption that this local weakness is necessarily due to an endocrine fault, nor does it warrant the statement that it is due to certain definite dietetic errors which, if corrected on the declaration of such weaknesses, would lead to their cure and which if corrected at an earlier date would prevent their occurrence.

I am inclined to think that, apart from endocrine errors and failures, and the bacterial toxæmia which is often the proximal cause of a breakdown, the vulnerability of various tissues is determined by biochemical faults, either of deprivation or of excess, which can be corrected by treatment on chemical lines. Such treatment must, of course, be combined with a dietary, not fixed either by general prejudice or by the ill-considered success of personal treatment, but by the careful study of the needs of each individual patient from the endocrine and biochemical point of view, due consideration being had with regard to any gross lesions which may be present. We know, for example, that all rheumatic affections are not due to the same cause, and that very similar rheumatic affections may attack people of quite different constitutions as estimated both from the endocrine and biochemical standpoint. We also know that, while a strongly alkaline water will help in one case it may be destructive in another, that one group of cases is best dealt with by sulphur water, that another does best on water containing iodine.

One thing to be remembered in the study of this question, is that the breakdowns of middle-age are due to failures of one kind or another, and it is important in all such breakdowns to see that elimination is adequate to tissue-change; to estimate the capacity of kidneys, skin and liver, and adapt the patient's life, dietetic, occupational and climatic, to suit that capacity.

Climate may play a large part in the production of breakdowns in middle-age. People who ought to live on the highlands are condemned to drag out a weary existence in Torquay; others who long for the soft climate of the west of England cannot leave Margate, Cromer or Buxton, and suffer accordingly; the effect of tropical and subtropical climates is well known, if little understood.

It is no credit to us as medical men that so many breakdowns occur in middle life. We are more occupied in attempting to "cure" a disease than in preventing it. Breakdowns of middle life due to the overstrain of a normal body are beyond the power of the physician to prevent, but those occurring without such abnormal strain are preventable in very many cases by the recognition in early life of constitutional tendencies which indicate either a biochemical or endocrine abnormality. All are not preventable, but if more attention were paid by the school doctor to the health of the slightly abnormal child or adolescent he would have fewer cases of what he recognizes as real diseases. Many breakdowns could have been prevented by a little extra care after measles or influenza occurring at school age, or by a little interest taken in a boy who felt sick or faint after exercise, but who was pronounced "all right" because no cardiac murmur or irregularity was found at the time of the medical examination. House masters are also greatly to blame who suspect that all complaints about health which are not associated with some easily detectable gross

physical lesion, are due to the desire of the boy to shirk some disagreeable thing in the way of exercise. Perfectly healthy boys love exercise.

To take an example, it is not unusual to find a man in early middle age, the subject of pulmonary tuberculosis, giving a story of ill-health of only a few months' duration. When the history is taken, however, it becomes clear that in this man's early life there was a period during which he was just ailing, when he "outgrew his strength" or had been subject to colds in winter that "went to his chest." He survived this period without developing any typical disease, and for, say, twenty years afterwards he remained well. Then for the first time he felt ill and was recognized either as a bad bronchitic or possibly a case of pulmonary tuberculosis. Physical examination reveals the presence of creaking, coarse rhonchi, and perhaps one or more small areas, not basal, of fine crepitations, and the X-ray picture reveals the past history of the lungs, dense fibrosis and more recent cloudy shadows. The X-ray picture makes it impossible to doubt that the fibrosis is old, and a study of the X-ray picture, the physical signs and the history makes it certain that the foundation of this breakdown of middle-age was laid in early youth. Had this man's lungs and constitution been studied and treated carefully during his earlier period of ill-health, it is most likely that the later breakdown would have been prevented. The recognition in early life of a very similar constitutional fault would prevent many of those most troublesome neurasthenias and cardio-vascular disorders of middle-age, especially those associated with gastro-intestinal disturbances, due to vagus failure. Those last are very important of recognition, as they can usually be cured at almost any stage.

In opening this discussion, I have merely endeavoured to show that these breakdowns are complicated events, involving questions of general resistance and local vulnerability, biochemical and endocrine abnormalities, dietetic and climatic errors, stupidity and carelessness, both of patients and of medical men. Also that are possible of prevention more than of cure, while at the same time the conditions co-operating in their production are more often than not capable of satisfactory modification if the conditions are not allowed to drift on too long.

Dr. R. Wybauw (Spa, Belgium).

Amongst the troubles of middle age for which we balneologists are most generally consulted, hyperpiesia takes a very important place. The reason is that in this disease, whilst it can be treated partly at home by diet, drugs give very little help; hydropathic or balneological treatment, however, yields good results.

Our ordinary explanations of the benefit obtained by this kind of patient at our spas are too superficial. We speak of "calming the heart," and of "lowering of the blood-pressure," expressions that mean little, and even the explanations for the mechanism of high blood-pressure that we ourselves accept are certainly insufficient.

Up till now, the theory has been that the heart is the only agent in the circulation of the blood, bringing the whole of it into movement without any other assistance.. The arteries have been considered as elastic tubes, nothing more, which, by keeping themselves more or less contracted, regulate the blood-flow to the irrigated organ.

If, in middle age, so many patients have an increased blood-pressure, the cause *must* be that the resistance to the outflow, on the side of the small arteries and capillaries is too strong; hyperpiesia is produced by contraction or spasm of the small arteries. In the great majority of our hyperpietic patients we find a high systolic

pressure, but the diastolic pressure is, in a very great number of cases, normal or only slightly increased.

If we try to imitate high pressure in a system of glass tubes through which we pass a rhythmically, intermittently flowing stream of water, we notice that every increase of the resistance to the outflow causes an increase of systolic as well as of diastolic pressure. Diastolic pressure always increases at a greater rate than the systolic. If the high tension of hyperpietic patients is caused by an increase of resistance in the small arteries, then we ought always to find the diastolic pressure distinctly raised. In practice, we usually find the contrary.

Hasebroek, of Hamburg, has written a good deal on the supposed systolic action of the arteries. His view is that, immediately after an artery has been distended by the pulse-wave there is an active reflex contraction of the arterial wall pressing on the blood. All the various parts of the arteries are subsequently excited, all contract one after the other, and this causes something like a peristaltic compression running along the arteries of the whole body, immediately following the pulse-wave.

The question was investigated by several German, Swiss and Czecho-Slovakian physiologists, and they obtained mostly negative results. It seems probable that the active movements of the arterial wall are very difficult to detect, because they appear so shortly after the greatest distension of the artery, so that it is quite impossible to differentiate them ; also, these movements are so weak in normal arteries that no method can detect them directly.

Several writers showed that when artificial circulation is established through an organ, the quantity of fluid passing through is larger when the flow of the stream is rhythmic than when it is continuous. In their experiments the rhythmic stream exercised a mean pressure equal to the pressure of the constant stream. The accurate experiments of Fleisch showed that this result is due only to the elasticity of the blood-vessels. No active collaboration of the arterial wall was required to explain the fact. We performed fresh experiments on the leg of a dog, on fresh kidneys, on human placental arteries, on the carotid arteries of sheep, and we found that when the stream was rhythmic these organs yielded a greater outflow than under a constant stream, even when the mean pressure was decidedly lower than the pressure of the constant stream. In that case, mere elasticity could not be regarded as the cause of the phenomenon, and we accepted the result as being due to an active response to pressure by the arterial wall. It is remarkable to see how a placental artery can increase the volume of a blood-stream when it is rhythmic. This artery is the one of which the muscular coat is the strongest of all.

We also made direct experiments on arteries, on the aorta of dogs and on carotid arteries of sheep shortly after death ; the arteries were submitted to the action of a rhythmic stream, and put in a kind of plethysmograph communicating with a Marey's recording cylinder. The movements of the artery when opened by the systolic wave, then kept open by the systolic pressure in our tubes, and during the collapse when the stream was cut off, were inscribed on the cylinder. When we first examine what happens if, instead of an artery, we insert a very elastic rubber tube into our plethysmograph, we see that, when the pulse wave reaches the tube, it opens suddenly ; this strong positive wave is followed by a short negative wave, due to a short elastic reaction of the wall. Then the tube keeps open as long as the high pressure lasts. When we put in the aorta instead of our rubber tube, we obtain an identical curve. If there is an active systolic reaction, this must occur together with the small negative wave. In order to ascertain whether this was really the case, we added adrenalin to our fluid (Locke's oxygenated and warmed solution). After a short time the negative wave became stronger and stronger, and a very marked active

reaction of the arterial wall was noticed. On the other hand, when we replaced adrenalin by a weak solution of morphine, the negative wave quite disappeared : this drug had stopped any active reaction in the arterial wall. In carotid arteries the reaction to adrenalin was not so positive, but the details of our tracings, which were taken at a very low pressure of the irrigating fluid, indicated a strongly developed response of the arterial wall to the pulse wave.

After having shown that high blood-pressure is not to be explained by spasm of the arteries, Hasebroek connected a water reservoir with a system of glass tubes and rubber tubes, and applied an apparatus resembling a cuff to a part of his rubber tube, somewhat similar to those cuffs we use in measuring blood-pressure but filled with water. He constructed his apparatus in such a way that, immediately after the rhythmic pulse began, a compression was made on the rubber tube. This was comparable to the effect of a contraction of the arterial wall directly following the onset of the pulse wave. He was able in this way, and in this way only, to increase the systolic blood-pressure, without changing the diastolic pressure ; by regulating the external compression he succeeded in regulating the high systolic pressure at will.

Let us now consider the state of our patients. It seems probable that hyperpietic patients have an arterial wall that responds too strongly to the excitation caused by the sudden shock of the pulse wave. There are reasons for supposing that their arteries indeed are too sensitive to any kind of excitation. It is known that in normal men or in normal animals the blood-pressure is the same in the arms as in the legs, but in many there is a tendency to a slightly higher pressure in the legs. In hyperpietic patients the difference is very frequently a great one ; there is some relative hypertension in the legs.

The experiments made by Pezzi, Fulchiero and Lusiada, have shown that, when the blood-pressure is taken first at the forearm, and then another cuff is applied to the arm, a pressure just a little higher than the diastolic pressure is indicated, while the blood-pressure in the forearm increases. We have verified this and find it to be rare in normal persons ; but it is the rule in hyperpietic patients. The cause of the phenomenon is that when compressed by a pressure just a little above diastolic pressure, the artery collapses during diastole ; immediately afterwards it is violently distended by the pulse ; and this violent shock seems to be an excitant to the reaction of the wall, but more so in hyperpietic people than in normal, because their arteries react too strongly.

We may therefore consider hyperpiesia as a state in which the local (probably nervous) reactions of the arterial wall are too strong, because there is some hyperæsthesia of its nerves.

There may be another cause of increased response of the artery, not arising from hyperæsthesia of the nerves. This may occur when the shock of the pulse wave is too strong,—in aortic incompetence, for example. There we generally find the blood-pressure in the legs much increased compared with the pressure in the brachial artery, a fact which was pointed out by Hill and Flack years ago, and which is very difficult to understand if the action of the arteries is similar to that of india-rubber tubes.

Danze, Brody and Miles last year (*Proceedings of the Society of Experimental Biology and Medicine*, 1926, vol. xxiii, p. 454) published a paper on pressor substances found in the blood of hyperpietic subjects. Do these substances act by themselves, or do they activate some internal secretions, like surrenal secretions, which cause hypertension ? After the experiments we have carried out, we think the unknown pressor substance does not itself act directly on the arterial wall, but causes hyperæsthesia of the arterial nervous mechanism.

What have these facts to do with our special balneological work? The connexion is indicated as follows:—

Our treatment has twofold effects. Hyperpietic (and often gouty) patients are poisoned by bad hygienic conditions and too rich food. At home, dietetic treatment may reduce their blood-pressure to a certain extent, but the only way further to reduce blood-pressure is by balneological treatment. Drugs are of but little use except in the treatment of some complications.

Balneological treatment is effective in such cases, first, because it acts on the metabolism, especially upon the metabolism of nitrogen; and secondly, because it has a calming effect on the nervous system, and soothes the irritability of blood-vessels. We see the difference between systolic and diastolic pressures diminishing after a fortnight, and after a month's treatment we generally obtain a lasting result.

Our idea (purely hypothetical) is that the reflexes in the artery are under control of the same nervous influences as the heart. Several experiments which we have made seem not only to indicate that the arterial wall is able to contract or to dilate,—as has always been accepted, but also that the immediate reflexes we have mentioned depend on exciting and moderating nervous influences. Nerves somewhat resembling the accelerans and vagus nerves appear to have their endings in the arterial muscles. And why not? Is not the heart a part of a differentiated blood-vessel, and do we not know of a vertebrate, the amphioxus, which lives without a heart, and upon whose blood-vessels alone devolves the control of the whole of the circulation of the blood? Our patients with heart trouble seem to be strengthened because the treatments act on the tonus of their vagus nerve; perhaps the same occurs in the blood-vessels of hyperpietic patients.

In any case, we see that the treatment of such patients has a more complicated effect than we hitherto supposed. The balneological "cures" act on the nervous system, on the heart and its nerves, on the vessels and their nerves, perhaps on internal secretion, certainly on metabolism. That is the reason why we cannot ascertain the result obtained when the patient is leaving the spa, and why so often the complete effect of a treatment does not appear for some weeks afterwards.

Dr. R. Fortescue Fox.

In an address delivered before the Hunterian Society[1] in 1922, the breakdowns of middle life were described primarily as a disturbance of all the great systemic functions, in varying degrees, which might subside, or lead on to anatomical changes in particular tissues or organs.

The analysis of many cases, at present unpublished, appears to confirm this hypothesis. If it proves correct it must modify the current views upon the ætiology and treatment of many disorders and so-called organic diseases in middle life.

The functions, in one or more of which the first signs of disorder are manifest, are naturally in order as follows: Mental, nervous, circulatory (cardio-vascular), metabolic (including endocrine function, digestion and assimilation), the eliminatory functions, and lastly, the functions of the locomotive organs (muscles and joints). The dependence of all these activities one upon another is obvious. If, for example, a disorder arises in elimination or assimilation, toxic matter or malnutrition may supervene in the whole body. Similarly, an endocrine disturbance speedily causes mental, nervous, circulatory, and digestive disorders. The same effect of disturbance of one function upon all the others is seen in most mental disorders. In fact, so

[1] *Lancet*, 1923, (i), 55.

26 Fortescue Fox: *Breakdowns of Middle Life*

closely are the great organic systems related that it seems impossible in many cases to decide in which of them some obvious disturbance has originated. In the common cardio-vascular diseases of middle life, especially those accompanied by hypertension and hyperpiesia, the circulatory disturbance is generally a secondary effect. Similarly, it is questionable how far the nervous centres are responsible for the great majority of nervous disorders. It is in the nature of these centres that their normal or abnormal activity should be evoked by external causes; that is to say in the afferent nerves or in the blood-stream.

According to this view the breakdowns of middle life should be regarded as general and not local diseases, and as probably connected with changes in the fluids of the body. The disturbance can be detected over a wide field, and even when one function or organ seems to be the chief sufferer, there may be others quite as much affected, and which may even be primarily at fault. An anatomical-pathological diagnosis describes rather the results of disease, and may overlook these underlying widespread disturbances.

To what causal factors may these widespread disorders of health in middle life be ascribed? Their incidence is heavy in the decade 45 to 55. This is generally recognized to be a period of physical involution and adjustment both in men and women. It is also the period of maximum strain, both physical and mental. Like the phases of adolescence and senility, the middle epoch of life has its own peculiar liabilities in normal health, and it is not unreasonable to believe that it influences any processes of disease which may take place while it lasts. Happily, it also gives them a transitory character. After the causal factors, epoch and strain, come injurious habits: unsuitable food, stimulants and narcotics, accumulation of waste, want of exercise and oxidation, often occurring together. Another serious factor cannot be overlooked—accidental invasion of micro-organisms, which become pathogenic on this well prepared field of lost immunities to disease.

In the address already mentioned, certain common types of illness in middle life are described and illustrated, in which the predominant symptoms are respectively mental, cardio-vascular, thyroid, arthritic, etc. Family histories often show similar forms of illness recurring in successive generations. This is especially true of the illnesses of middle life and perhaps of the adolescent and the senile periods also. For example, arterial disease with cerebral hæmorrhage at 50 or 60 years of age (death at the same age being not uncommon), mental breakdowns at the climacteric period, the less acute forms of arthritis and many forms of chronic rheumatism, are met with in certain families much more frequently than in others.

From such observations as these it may be legitimately deduced that family tendency is a chief factor in determining the localization of disease in middle life. A general disturbance of the equilibrium of health, with disorders of many functions due to epoch, strain, injurious habits and micro-organisms, leads on naturally to more serious and permanent disorders and tissue change in the most susceptible parts. For this reason the investigation of family history in middle life has much more than academic interest. It is essential that it should be undertaken in order to comprehend the morbid liabilities of this period, and above all to provide an effectual preventive treatment. It is, of course, a truism that in all the illnesses of middle life that can be characterized as "breakdowns," an escape, at least temporary, must be made from the conditions in which the illness originated. Change of habits, regulated rest and exercise and holidays, fasting, elimination of alcohol, saline and Turkish baths, are everyday prescriptions, sufficient for all the less serious cases of middle-age disturbance, mental and physical. But there is a very large and perhaps increasing number of cases in which these simple remedies are insufficient. In spite of the best treatment

at home, the patients often go from bad to worse and the general disturbance settles down into localized disease. Sleep or mental equilibrium fails, or the pulse remains too frequent; arterial hypertension, wasting or obesity are not checked; the skin, kidneys or bowels remain still inactive, or pains and swellings in and about the joints do not disappear. Careful rectification of possible sources of infection still leave these persons very far from well, with marked general disturbance of health and increasing signs of local disease.

From this class of case the health resorts draw large numbers of their more serious visitors. At many health resorts the tonic or sedative climate is itself a remedy of no slight importance, especially when combined with regulation of diet, rest and exercise in the open air. This is much, but the spas offer more—internal treatment by a variety of waters and also different forms of external treatment. Both at climatic stations and at spas, special sanatoria for more serious cases are, or might be, founded. A whole armoury of physical remedies applied under the most favourable conditions is therefore available in these health centres. Does it not follow that upon the practitioners at the spa, climatic station and sanatorium must devolve the care of this important class of case. Above all, the scientific hydrologist has the means at his command, not only of arousing dormant nervous reactions, but also of allaying excessive nervous or cardio-vascular excitation; of correcting defective elimination whether by bowel, kidneys or skin; of increasing oxidation and metabolism; of modifying chronic inflammatory processes and of dispersing morbid exudations and deposits.

The adjustment of these remedies of the spa and health resort to the diseases of middle life is a very fine art. If the spa practitioner could study the circulatory and nervous reactions evoked by baths,—the external treatment of sensitive subjects, treatments could sometimes be modified from day to day with much advantage. Many patients require the most sedative applications that can be devised, others need a daily increasing stimulus. Like strychnine, bromides and arsenic, baths and waters are edged tools, and their selection and dosage must be guided in every case by the reactions observed. This is more especially true with the varied and intricate disturbances of health occurring in middle life.

Dr. W. J. Tyson (Folkestone).

The breakdowns of middle life depend largely on the bringing up of the child. Provided that the child is well cared for as regards environment—feeding and clothing, and that this treatment is carried out up to adolescence, there will be few breakdowns in middle life; if when this time has arrived common-sense rules relating to feeding, exercise, moderate work, etc. are observed—old age can be reached and enjoyed. It is impossible in a few remarks to mention, far less describe, all the details of these rules, but these need not be many nor need they be complicated.

Dr. H. A. Ellis.

How one approaches middle life is largely a constitutional question. It depends to a great extent on whether during earlier years we have (*a*) been normal individuals, whether (*b*) we have had a difficulty of assimilation and have therefore been subject to metabolic deficiency, or whether (*c*) we have already shown a tendency to deficient elimination. Taking these as the three types of constitution one can definitely say that those who early display difficulties in elimination are subject to premature

28 Ellis: *Breakdowns of Middle Life*

age, are liable to cardio-renal disorders in early middle life and that they have an unhealthy future in front of them.

It must be remembered that life is an acid phenomenon in an alkaline or neutral medium, and that age has a tendency to increase that acidity at the expense of the environmental alkalinity. So we have these three types of constitution and these types definitely control middle-age conditions and the ailments of the individuals approaching that period. These three types may be called (1) the acid or assimilation error type, (2) the normal or balanced type, and (3) the elimination error type. The urinary excretions of these three types are totally different and capable of accurate recognition when their elimination and acidity are considered. Acid is eliminated in the urine in two ways, (*a*) as free acid and (*b*) as salts, when the acid is combined with ammonia.

Now the ratio of these two to each other differentiates the constitution in its acid relations, and that differentiation will reveal the acid characteristics likely to occur in middle life. In the ordinary healthy individual the proportion of ammonia to free acid is in the neighbourhood of 2 to 1, or the average ratio. Those, however, who encounter metabolic strain and elimination difficulties will have a reducing ratio till they arrive at the 1 to 1 ratio. This is the acid type, with elimination difficulties, that forms such a large proportion of those who consult us for disabilities in middle life. These patients essentially tend to become cardio-renal cases with a rising blood-pressure and an increased differential pulse due to the need of getting rid of their accumulated waste acid, and their diminished capacity for its elimination. It must be understood that acid is the main controller of the kidney function, so a deficient capacity for acid elimination on the part of the kidney is the chief reason for the onset of cardio-renal deteriorations and disturbances. The acids formed by vital processes must be eliminated or their accumulation will cause degenerative disturbances on an accelerating plane. The question is, how these disturbances can early be recognized and obviated. There are three ways by which waste accumulation can early be recognized: (1) By observing the *rise of blood-pressure and increase* of differential pulse-pressure. This indicates the deterioration of the renal *elimination function*. This deterioration necessitates an extra pressure in order to facilitate the filtration of the excessive waste products. (2) By *investigation of night micturition*. The most important consideration for middle age is, do the patients have to pass water at night, and if so, how often? That also usually co-ordinates with kidney deterioration, A man is not really as old as his arteries, but as old as his kidney capacity, which controls the deterioration of the arterial walls. (3) *By urinary analysis*, the earliest and best way. For this two samples are necessary, the one passed on waking after a night's sleep, secreted under the metabolic conditions of rest, equable temperature, and absence of food and drink. This may be called the standard or rest urine. The second specimen is the next one passed, that is the first one secreted after waking in the morning and passed, say, after breakfast. In normal individuals, if sleep has been sufficient and the kidneys are unimpaired, all the accumulation of free acid has been excreted during the night. Then this second urine will have little or no acid in it. In the average normal individual its specific gravity will drop about 10 degress lower. The

reaction will be probably alkaline. This second urine may be called the tide urine. It shows to what extent elimination is complete in an acid-accumulating case. With a deteriorating kidney the tide differences will be much smaller or will have disappeared altogether. So the two specimens, instead of being separated by a specific gravity of 10 degrees, will be approximately equal. The acid also will not be reduced; the tide urine will approximate to the rest urine, showing that accumulation is occurring and the night elimination has not been sufficient to get rid of the waste. The blood-pressure and night micturition will further confirm this. As the case retrogrades the night specific gravity tends to become lower and the tide to become higher until the difference between them entirely disappears.

If, on the other hand, the case belongs to the assimilation error group these disadvantages cannot occur, as, where there is not sufficient assimilation, there cannot be difficulties of elimination.

These facts show the way to treatment in acid cases, which will naturally consist in controlling waste formation, more than in aiding accumulation, if the age clock is to be definitely and satisfactorily put back. Increasing elimination is only a palliative treatment, very efficient and satisfactory if the deterioration has not gone too far; this is duly apparent from the improvement achieved in the various watering places where the physicians have studied the elimination side of the problem to its scientific limit, and where the results are practically excellent. But if the condition is not to recur the waste production must be controlled either by diminishing intake or diminishing work, and this can only be satisfactorily accomplished by studying the urinary results to see how far the state of the tide urine can be improved by diminishing the amount of acidity which is found in it; it will then co-ordinate with a diminished blood-pressure and longer periods of undisturbed sleep.

In the other group of cases, that is, those in which there are assimilation errors in youth and in which there is an ammonia ratio of 3 to 1 in relation to the free acid, the patients grow old quite differently. Should they deteriorate in middle age, their error will be found to be one of the endocrine balance, this being naturally deficient. These patients have a low blood-pressure, a differential pulse-pressure below 44, and they do not pass water during the night. They also have a good tide. In other words they have no acid accumulation, but their endocrines tend to fail; hence their fatigue and other disabilities.

A few words on endocrine failure may be interesting. Endocrines do not fail all equally, nor altogether, so it is of great importance to find out the one that deteriorates first, for if it can be replaced by a suitable dose the endocrine balance can be re-established and all will be well.

One word of warning: endocrines are usually not needed in the acid constitution with elimination errors, as, naturally, if elimination is already deficient no further accentuation of this condition is advisable or beneficial. Indeed, if the patient's health does improve temporarily, that may lead to a still more serious breakdown.

In this connexion, recently, in a case in which ill-health followed the administration of small doses of parathyroid I found that the cause of the ill-health was an acidosis thereby produced, and the pH fell very nearly to 72, there being also a considerable deficiency of the bicarbonate reserve. This was remedied by giving 10 gr. of sodium bicarbonate

whenever a dose of thyroid was administered, as the thyroid deficiency still existed, and the condition of deterioration was successfully remedied by giving the sodium bicarbonate as well.

These observations I hope will render possible a discussion as to how far middle-age deterioration is, as the opener pointed out, a constitutional biochemical and endocrine question.

	Rest	Tide
Normal Case.		
Titratable acidity	26	4
NH_3, combined acid	48	25
Normal Acid Case.		
Titratable acidity	38	8
NH_3, combined acid	48	12
Acid Case.		
Titratable acidity	45	31
NH_3, combined acid	54	43
Normal Alkaline Case.		
Titratable acidity	12	6
NH_3, combined acid	27	20

Dr. Mougeot (Royat).

I will briefly discuss the question of high blood-pressure on balneological treatment. Dr. Wybauw has shown the part played by increased reflex contractility of the arteries of muscular types. I think the cause of that resides in increased tonicity of the sympathetic system. This system has a large domain under control, many parts of which contribute to the production of hyperpiesis, increasing the activity of the heart muscle, and also the tonicity in the arterioles and the capillaries.

Krogh has shown that capillary tonus is regulated by the pituitary secretion and the sympathetic nerves. I think that both carbonic-acid baths and also radio-active hyperthermal baths reduce the high blood-pressure, by their soothing effect on the sympathetic system; and that amongst the different kinds of effervescent baths the most efficient are the naturally thermal waters with a weak amount of CO_2 and radio-active properties.

Dr. Wm. P. Kennedy (Bath)

said he thought that monotony in dietary, even when controlled in quantity, deserved a place in the consideration of the influences affecting the breakdowns of middle life. It was not so important as the subject of over-feeding, but it was an aspect of the question one could not afford to neglect.

Mr. H. L. Attwater.

Speaking as a surgeon I would say that the urinary passages show a remarkable power of resisting organisms; this is demonstrated by the frequent presence of bacteria in the bladder, which are derived either from the urethra below or from the

kidneys above, and which do not cause any disease or disability. Further, if there is an infection, following upon some primary source of irritation, such as a vesical stone, this will most frequently disappear rapidly when the source of irritation is removed.

In middle age, however, when there is a general slackness of the abdominal and lumbar muscles, which is due to a fatty infiltration and a loss of tone, the urinary passages are subjected to less vigorous disturbance from without; the patient's condition becomes more sluggish, and there is a much greater tendency to stagnation and to a less rapid clearance of the urine from the channels. This favours stasis; infections become much more common and lead to one of the forms of breakdown in middle life. Spa treatment in such cases is very valuable. The imbibition of large quantities of diuretic waters, such as those of Contrexéville or Vichy, causes the urine to pass more quickly along the canals and will tend both to prevent and to check the onset of sepsis.

With regard to the remarks of Dr. Ellis, with whom I have been associated during the last two years, whilst he sees a majority of nephritic cases which belong to the "acid" group, the cases which I see of infections of the urinary passages belong to the "alkaline" group. "Acid" cases are exceptional.

Dr. Paul Ferreyrolles (La Bourboule).

There is something more in the mineral spas than régime, rest, exercise and fresh air, to enable patients to fight against the miseries of middle age; there is the mineral water itself.

As Dr. McClure has rightly remarked, the clogging of the human machine is perhaps the result of our own misdeeds, but also of our previous illnesses, which have left our organism filled with toxins. It is well to increase their elimination, and to diminish their absorption, but it is best to complete their destruction. I have had the opportunity, on a previous occasion, of telling you something about the anti-anaphylactic power of some mineral waters.

Certain of them act not only on the organism but also on the toxin itself. This has been recently proved by the eminent physiologist, Professor Billard, who has shown that the waters of Mont Dore and of La Bourboule destroy the toxic power of spartein; that the toxic action of phallin of certain fungi is destroyed by the Source des Chanteurs at Mont Dore; that adder venom is destroyed by the water of Châtel-Guyon; the diphtheria toxin by that of Saint-Nectaire, and the poison of tetanus by the water of La Bourboule, and in a lesser degree by that of Royat.

You all know the extraordinary influence on the phenomena of life played by certain ions in infinitesimal doses. This has been shown by Raulin in the use of silver and zinc salts, by Bertrand working with manganese, and by Bertrand and Macheboeuf employing nickel and cobalt.

Realize that you can find more than sixty (component) minerals in Vichy water; that if the intoxication of middle age is complex the mineral waters also are

complex, and that you can find in them, and in them alone, the ions or groups of ions, able to neutralize these toxins. It is to the effect of these ions that we attribute the results of spa treatment. I earnestly recommend you to study the experiments of Professor Billard and to repeat his experiments with your own waters. You will then be armed against the polite scepticism of some general practitioners and patients and be enabled to say why you believe in the efficacy of mineral waters.

Clinical Section.

President—Sir HERBERT WATERHOUSE, F.R.C.S.

CASES.

Chronic Renal Disease with Bony Changes.

By A. A. OSMAN, M.R.C.P.Lond.

H. P., AGED 12. First complained of weakness of legs 5 to 6 years ago. Attended Out-Patients' Department August 25, 1926. Thin, pale, and undersized, with marked genu valgum.

Urine then contained albumin, 1 part per 1,000, a good trace of sugar, a few red cells, and epithelial cells in the urine, but no casts. Later, a few hyaline casts were observed.

Heart and arteries normal. Systolic blood-pressure normal, = 90 mm. Hg. Retinæ normal. Wassermann reaction negative.

Blood-count: red blood-cells, 4,112,000; white blood-cells, 8,000; Hb. 70 per cent.; colour index, 0·9; polymorphonuclears 58 per cent., lymphocytes 38 per cent., eosinophils 4 per cent.; plasma $NaHCO_3$, 0·025 (normal 0·032 molar); blood urea, 21 mgm. per 100 c.c.; blood calcium, 10 mgm. per 100 c.c.

Urea concentration:—

	VOL.		C.C.N.		
First hour	52 c.c.	...	40·2 c.c.	...	2·51 per cent. urea
Second ,,	40 c.c.	...	60·2 c.c.	...	3·76 ,, ,,
Third ,,	36 c.c.	...	60·5 c.c.	...	3·78 ,, ,,

Repeated examinations of the urine for tubercle bacilli proved negative. There were no signs of tuberculosis in the chest that could be detected clinically or by X-ray. Radiograms of the kidney regions showed shadows suggesting calcified glands in the region of the left kidney. A pyelograph by Mr. J. Alban Andrews showed that these shadows were not in the kidney. The temperature charts shown suggest tuberculosis.

After some weeks' treatment with alkalies the albuminuria and glycosuria disappeared. The X-ray appearances of the bones are not typical of renal dwarfism, or late rickets. The case is shown for opinions as to the possible connexion, if any, between the bony and renal lesions.

Case for Diagnosis.

By A. A. OSMAN, M.R.C.P.Lond.

V. L., AGED 3½. An only child, born at full-time, thirteen years after the only other pregnancy, which resulted in a miscarriage. Breast-fed for one year and three months. At this age attended Out-Patients' Department, for diarrhœa of a few days'

duration. Child pale and thin, but not very ill. There was a bilateral, symmetrical swelling of the malar bones and slight beading of the ribs. No other signs of rickets. Up to this date patient's development had been normal. Wassermann reaction negative in both mother and child. Patient has attended Out-Patients' Department since at regular intervals, and during this time the bony swelling over the left malar region has slowly increased in size, causing a slight but definite proptosis of the left eye. The swelling over the right malar region has not increased in size, and possibly it is even smaller than when first seen. X-ray examination of the skull gives no information as to the nature of the swelling. The urine contains no abnormal constituents.

Dr. F. PARKES WEBER thought the bony swelling on the left side of the skull was allied to what was formerly called leontiasis ossea, and was due to a condition of osteitis fibrosa.

Three Cases of Arthroplasty for Ankylosis of the Temporo-Mandibular Joint.

By E. C. HUGHES, O.B.E., M.Ch.

Case I.—W. R., male, aged 10. In May, 1921, the right side of his face became swollen and two teeth were extracted. The swelling did not subside, and in the following August an incision was made on the inner side of his cheek and non-purulent serous fluid evacuated. A month later two lower milk molars were extracted and both the external and internal alveolar margins were found to be necrosed. They were removed. After that there was very little discharge, but there was a continuous offensive smell. Later, the right side of the face again became swollen from bony overgrowth of the mandible.

In June, 1922, a sequestrum was removed. In October of the same year he was again admitted for disability of mastication. Another sequestrum was removed. The lower jaw was then slightly deviated to the right, and the left side was not well developed. In December of the same year the articular process of the lower jaw on the right side became loose and was removed.

In June, 1926, he was re-admitted into hospital for ankylosis of the right temporo-mandibular joint. The new bone, condyle, and coronoid process were removed.

Case II.—D. W., female, aged 11. Fixation and immobility of the lower jaw noticed by the mother three weeks after birth. There was no history of accident.

Patient admitted to Guy's Hospital at the age of 4 in June, 1919, when it was noticed that she had no chin, but enlargement and bulging of the right cheek with flatness of the left. The mandible was deviated towards the right. The lower teeth sloped backwards, and between the upper and lower rows there was an interval of about $\frac{1}{2}$ in. There was absolute fixation of the lower jaw; patient had never eaten any solid food and her power of speech was very poor.

The right joint was then operated upon and the neck of the mandible was found to be short and thick. It expanded into the condyle without the definite thinning occurring normally. The condyle was flattened rather than rounded. Between it and the fossa no trace of the articular cartilage was found, but there was a dense band of fibrous tissue. This was removed, together with the condyle and part of the neck. A strip of temporal fascia was then inserted between the fossa and the stump of the neck of the mandible. A week later the left side was opened, but the joint was found to be normal.

Case III.—R. M., male, aged 18. Patient said he was knocked over by a bicycle

about fourteen years ago and both his skull and mandible were fractured. Ever since then he has been unable to open his mouth.

In February of this year he was admitted to Guy's Hospital, and he could then only depress his mandible about ¼ in. The mandible was retracted about ½ in. behind the maxilla, and there appeared to be considerable thickening in the region of the temporo-mandibular joints on both sides. X-ray examination failed to reveal the site of the old fracture, though it appeared that the right joint was more seriously affected than the left. The right side was therefore opened first, and when the joint was exposed firm, bony ankylosis was found. The condyle and part of the neck of the ascending ramus were removed so that a gap of about ¼ in. was left between the two bony surfaces. Into this a piece of temporal fascia was inserted. A week later the left side was opened and a similar, though less difficult, operation was performed.

Two dental props were then placed in the mouth in the molar region. At first these were left in altogether for two days. They were then removed for meals, and later throughout the day, movement of the lower jaw being encouraged by the use of chewing gum, etc. More recently patient has been fitted by the Dental Department with a special prop to wear at night.

On February 21 he could separate his teeth sufficiently well to enable him to push his tongue between them, and the range of movement is steadily increasing. There is still, however, some resistance on the right side which may need further operative treatment.

Syringomyelia with Charcot's Shoulder.

By C. P. Symonds, M.D.

G. B., aged 27, male. First attended hospital in May, 1926, on account of pain and swelling in the right shoulder.

His story was that two days before, on a Sunday evening, he had felt a sharp pain on the right side of his neck, which ran down into his shoulder; subsequently there was swelling of the shoulder and some pain on movement, but he went to work as usual on the following day.

On examination there was found to be great swelling of the right shoulder, apparently due to distension of the shoulder-joint, together with compression of the veins; for there was some œdema of the arm, extending down to the back of the hand. Active movement was limited, passive movement was free but somewhat painful, and was attended by marked crepitus in the joint.

X-ray examination showed: (1) extensive destruction of the upper end of the humerus, the head of the bone having completely disappeared, (2) a fracture of the greater tuberosity, and (3) new bone formation in the soft tissues.

Neurological examination showed an area of dissociated anæsthesia involving on the left side the fourth cervical root area, extending across the chest to the right shoulder and involving the cervical root areas 4 to 8 and the thoracic root area 1 on this side. The tendon jerks of both arms were absent and the plantar response from the right foot was extensor. Over these areas cotton-wool sensation was normally appreciated, the painful element of pin-prick was not felt, heat and cold were imperfectly distinguished, sense of position and passive moment were normal. There was no noticeable muscular wasting or weakness.

52 Saner: *Gastro-enterostomy;* Cassidy: *Achalazia*

Case of Gastro-enterostomy.

By F. D. SANER, F.R.C.S.

W. G., MALE, aged 50. The history of this case extends over the past twenty-four years, includes five major abdominal operations and an almost uninterrupted record of dyspeptic symptoms.

1907-1912.—Vague dyspeptic symptoms.

1912.—Acute suppurative appendicitis. Operation. Drainage.

States that after this dyspeptic symptoms became worse and he dates his troubles definitely from this time.

1913, June.—Admitted to Lewisham Infirmary with a perforated duodenal ulcer. Operation, closure, perforation and drainage.

1913, October.—Since previous operation had continued to suffer with dyspeptic symptoms. In October was re-admitted to Lewisham Infirmary and a gastro-enterostomy was performed.

1913-1924.—No definite improvement. Discomfort was experienced after any meal, and diet did not alter this. Never any actual pain.

1924, February.—Admitted to St. John's Hospital, Lewisham, under Dr. Jackson. Symptoms much the same as before, only increasing in intensity; especial complaint of pain in epigastrium and left lower chest.

X-ray Report (summary).—Pylorus closed. The opaque meal leaves by the gastro-enterostomy opening at a great rate, and within ten minutes is entirely collected in the small intestine. No evidence on which to base suspicion of jejunal ulcer.

Operation, Laparotomy (F.D.S.). Stomach rather low in position but good tone. Gastro-enterostomy opening easily admitted hand. Closed so as to admit two fingers. Pylorus patent.

1924, July.—No improvement in symptoms.

X-ray Report (summary).—Meal was found to have left stomach completely within five minutes. Laparotomy (F.D.S.). Gastro-enterostomy closed.

1924-1926.—Pain present every day for eighteen months. Relief obtained at various times during day but never for whole day. Improvement for six months (April to November, 1926). Freedom from pain for periods two to three weeks at a time. In November symptoms recommenced as before.

1927.—Pain became severe and more persistent; present at night; subject to exacerbations, but patient now never completely free from it. A tender "tumour" can now be palpated in epigastric region, a feature not previously noticed.

X-ray Report, January 14, 1927.—Stomach dilated and ptosed. Some pyloric and pre-pyloric deformity. Pylorus does not function well and appears constricted. Stomach takes over six hours to empty.

Case of Achalazia.

By MAURICE CASSIDY, M.D.

T. N., AGED 60, a printer's warehouseman, complains of difficulty in swallowing of about ten years' duration. He cannot remember whether his symptoms commenced abruptly or gradually, but thinks that they have not varied much since their onset. Immediately on swallowing any kind of food, solid or liquid, he experiences a sense of discomfort under the lower end of the sternum which rapidly merges into a pain of great severity between the shoulder blades; the pain often makes him writhe,

and continues for a short time until he regurgitates; regurgitation always relieves the pain completely. He does not think that he regurgitates the whole of the food swallowed. Recently the difficulty in swallowing has gradually increased, a larger proportion of the food swallowed being returned. He has lost between three and four stone in weight during the last four years.

On admission he was pale and emaciated, weighing only 7 st. 2 lb. in a dressing gown. On examination of the chest and abdomen nothing abnormal was detected except a little bronchitis. X-ray examination revealed an enormous dilatation of the whole œsophagus due to obstruction at the cardiac orifice. The Wassermann reaction was negative; examination of the œsophageal contents, and of the resting gastric juice, and of the juice one hour after a meal, revealed absence of free hydrochloric acid in each instance. A mercury bougie was introduced into the œsophagus and it passed without any difficulty; the results were unsatisfactory, however, at first because, as proved radiographically, on many occasions the bougie did not enter the stomach but engaged in a pocket at the dilated lower end of the œsophagus.

On January 28 œsophagoscopy was performed by Mr. Howarth, who dilated his cardiac orifice by means of a special metal expanding dilator. On February 12 he was put on inj. atropine, $\frac{1}{100}$ gr. b.d., and luminal, $\frac{1}{2}$ gr. o.n.

As a result of two months' treatment the patient has improved considerably and gained 14 lb. in weight. He never regurgitates more than twice a day, often only once; the amount regurgitated rarely exceeds half a teacupful. He continues to pass the mercury bougie before each feed. Advice is asked as to the future treatment of this case.

Dr. CASSIDY said that this seemed to be a straightforward case of achalazia which after some initial difficulties had progressed very satisfactorily up to a point. Though there was an enormous improvement in the general condition of the patient, who could now swallow a fluid diet easily after passing a mercury bougie, solid food could not be taken, and he (Dr. Cassidy) doubted whether the patient would be able to dispense with the bougie. He was not inclined to advise surgical treatment. Walton had reported a successful series of cases treated by digital dilatation of the cardia after gastrostomy, but in two cases at St. Thomas's Hospital so treated by Maybury and Howarth the results had not been satisfactory. Œsophagogastrostomy, and the operation proposed by Rowlands based on Rammstedt's operation, had also been considered.

By the kindness of Dr. Adrian Stokes the Section had had the opportunity of seeing the very beautiful and convincing histological preparations prepared by Mr. Rakes, of Guy's Hospital. Dr. Cassidy thought that those who had seen these slides would agree that Mr. Rakes had established a morbid anatomy for this condition where previously there had been no recognized pathological basis.

Two Cases of Madelung's Deformity.

By C. C. LAMBRINUDI, F.R.C.S.

I. AND G. H., two sisters, aged 15 and 16 respectively.

Within six months of each other these patients were referred to the fracture department at Guy's Hospital by two different casualty officers, for pain and deformity of the wrist thought to have been produced by an injury.

On examination, there was marked deformity, the hand being carried forward, the ulnar styloid process was very prominent, there was some restriction of extension and an ill-defined feeling of weakness, insecurity and pain.

X-ray examination showed a forward curve of the lower end of the radius, a dislocation of the lower radio-ulnar joint, and an area of lessened density on the inner

aspect of the lower end of the radius; the epiphyseal line appeared to be indistinct anteriorly, as if prematurely fused.

Moore, writing in the *Journal of Bone and Joint Surgery*,[1] states that there are two classes of cases indiscriminately included under the title of Madelung's deformity. In Madelung's original description in 1878 the carpus was stated to be simply dislocated forward without any associated bone deformity. It was Duplay, in 1885, who drew attention to cases having a similar appearance, but with an associated anterior curve of the radius. This latter variety, of which my cases are an example, is very rare; according to Moore only seventy-five cases have been recorded.

It is commoner in girls, and starts between the ages of 12 to 16 with pain and deformity of the wrist. The pain, which may have been excited by occupation or slight trauma, subsides spontaneously, but the deformity gradually increases, and is presumably brought to an end by the fusion of the epiphysis with the diaphysis at the age of 20.

Such a deformed wrist with limited extension, even though painless, must always be a weak one, as the patient is unable comfortably to place the hand in the position which gives maximum power.

The ætiology of this deformity is obscure. I think the X-ray photograph of I.H. does show premature ossification of the anterior part of the epiphyseal cartilage.

Moore is inclined to class these cases in that ever-increasing group of epiphyseal abnormality of which Perthes' and Köhler's disease are familiar examples, but the cause is still undecided.

Intermittent Œdema of the Foot for Diagnosis.

By C. C. Worster-Drought, M.D.

F. L. J., aged 34.

History.—Enlisted 1916 and stood training well. After several "forced marches" in 1917 began to complain of excessive fatigue, but nevertheless proceeded to France in September, 1917. In January, 1918, he developed "P. U. O," and in May, 1918, was sent back to England with a damaged hand. Later in the same year he was discharged from the Army and remained well until September, 1920, when he began to complain of periodical "swelling" of the right foot. On the first occasion (September, 1920) the swelling lasted one month and did not recur until June, 1921, when it persisted for five months. During the past five years the intervals of remission have become progressively shorter until now, when some degree of œdema is almost always present.

No previous illnesses of importance.

Physical Signs and Progress.—When first seen in July, 1921, the patient presented the following signs: Moderate œdema of right foot, especially of dorsum, extending as far as the ankle and pitting deeply on pressure. Heart: No enlargement and sounds normal. State of arteries good. Pulse 112 rising to 128 on exertion-test, but falling again to 112 within a minute. Dorsalis-pedis and posterior-tibial pulses equal and normal. Pulsatile aorta; vasomotor instability. Liver and spleen not palpable.

Nervous system: Cranial nerves normal, apart from old strabismus; abdominal reflexes brisk and equal and deep reflexes normal.

The swelling of the foot disappeared after five months, but he was not seen again until November, 1921. There was then no œdema of the foot, but his hands were bright-red in colour, and he exhibited general vasomotor instability of cutaneous stimulation.

[1] *Journ. Bone and Joint Surg.*, July, 1924, 568.

The œdema remained absent until February, 1922, when he was again seen. Œdema was then present as before, and in addition there was marked cyanosis of the toes. He was treated with calcium lactate, gr. 10 t.d.s., until October, 1922, without benefit.

From December, 1922, to February, 1923, he received twelve intramuscular injections of "collosol calcium," 1 c.c., at weekly intervals. The œdema was absent for several days during January, 1923.

From February, 1923, to August, 1923, he received twenty-four intravenous injections of "afenil" (calcium-chloride-urea) at weekly intervals. The œdema was absent for short intervals, but no pronounced benefit resulted.

In October, 1923, his walking appeared more difficult, and there was a suggestion of slight spasticity of the right leg. The following physical signs were noted: Pupils and cranial nerves normal; arm-jerks moderate and equal; abdominal and epigastric reflexes absent; knee-jerks brisk, right slightly greater than left; ankle-jerks brisk, right ankle clonus; plantar reflexes flexor. Sensation everywhere normal; co-ordination normal. Other systems normal.

At no time did the urine show any albumin or casts. The blood yielded a negative Wassermann reaction, and the cerebro-spinal fluid showed a normal cell count and negative globulin, Wassermann and colloidal-gold reactions.

During 1925 he complained of occasional weakness of the right arm, but no abnormal signs were detected. At the present time, however, the right arm shows some general wasting as compared with the left ($\frac{3}{4}$ in. wasting forearm, and $\frac{1}{2}$ in. upper arm). Beyond the fact that the knee-jerks are rather brisker, his condition continues much the same. The right knee-jerk is brisker than the left; there is right ankle clonus and the abdominal reflexes remain absent. The plantar reflexes are variable, occasionally weakly extensor, but more often flexor.

COMMENTARY.

The original diagnosis, until the appearance of spasticity in the leg, was "angio-neurotic œdema." With the advent of signs suggesting the development of lateral sclerosis, the question of early syringomyelia was considered, the œdema being a possible, though rare, initial trophic change. Sensation to all forms of stimuli, however, have invariably been normal. At present, one is inclined to regard the case as a very slowly developing and somewhat abnormal form of amyotrophic lateral sclerosis, though even upon that supposition it is difficult to explain the appearance of intermittent œdema of the foot three years before any other sign.

Swelling of Finger.

By A. E. MORTIMER WOOLF, F.R.C.S.

SWELLING inside finger noticed since birth. Getting bigger lately; the mother thinks the swelling increased in ratio to the general growth.

On the palmar aspect of the middle finger (right hand) are two swellings situated on the proximal and intermediate phalanges. There is a smaller swelling on the dorsal aspect of the latter.

There is also a small subcutaneous swelling at the level of the insertion of the right deltoid.

Dr. PARKES WEBER thought the little nodules were neuromata of some kind. There were no molluscous fibromata or patches of cutaneous pigmentation to suggest Recklinghausen's disease.[1]

[1] *Corrigendum.*—These remarks were printed, by an error in the last number of the *Proceedings* (see No. 6, April, Clin. Sect. p. 42).

56 Burrell: *Pulmonary Tuberculosis;* Hare: *Scleroderma*

Neoplasm of Lung.

By L. S. T. Burrell, M.D.

Male, aged 52. Quite well three months ago when he began to suffer from dyspnœa. Cough for two months; patient was thought to have bronchitis. No pain. At present dulness all over left side of chest and breath sounds weak. Heart position normal.

Needle put into left side and was felt to pass through thick resistant matter. No fluid found. X-ray examination: Dense opacity over left side of chest.

Cases of Pulmonary Tuberculosis treated with Sanocrysin.

By L. S. T. Burrell, M.D.

Case I.—Male, aged 17. Onset of pulmonary tuberculosis July, 1926. In January, 1927, there was considerable infiltration of both apices, as shown in radiogram. Evening temperature 100° F. Tubercle bacilli numerous in the sputum. The chart shows the reactions to the sanocrysin. No tubercle bacilli in sputum in the last two examinations.

Case II.—Female, aged 28. Pulmonary tuberculosis developed in March, 1926. In December there was considerable disease, as shown in radiogram. No tubercle bacilli were found in the sputum in February after sanocrysin treatment. She had reactions as shown in the chart after two sanocrysin injections.

Two Cases of Scleroderma.

By D. C. Hare, C.B.E., M.D.

Case I.

Mrs. B. W., aged 40. Present trouble began about four and a half years ago when the patient had "rheumatism" in the joints, beginning in the fingers but also extending to the wrists, knees and ankles. It became much worse during 1926 when the hands were gradually "drawn up." Patient first seen in November, 1926, when the general condition was much as at present, with the addition of very severe oral sepsis.

Past History.—Nothing to note.

Present Condition.—Very thin; physique poor. *Face:* Skin thick, white and drawn tightly over the bones. Many telangiectases. Tongue protruded with difficulty. Voice hoarse and weak. *Hands:* Skin white and glossy, and sweating profusely. Fingers tapering, stiff and almost immobile, terminal phalanges displaced downwards. *Other Joints:* Wrists, shoulders and knees all stiff and the movements painful. Some grating on movement. The hips, elbows and spine appear to be unaffected.

Treatment.—By extraction of teeth and by administration of an autogenous vaccine from a septic root.

Clinical Section 57

CASE II.

SCLERODERMA WITH SUBCUTANEOUS NODULES.

MRS. H. P., aged 28. The patient was shown before this Section last session[1] and is shown again as then requested.

The present trouble began about six years ago and she first noticed the nodules about three years ago.

Past History.—Nothing to note. No septic focus ever found.

Present Condition.—The patient is fairly well nourished but of poor physique. *Face*: Nose slightly cyanosed and the skin shiny. The voice is hoarse but has been so for many years since an operation for removal of tonsils.

The Hands: Hands and forearms deeply cyanosed; skin atrophic and shiny and sweating noticeable. The fingers are stiff and are moved with difficulty. The terminal phalanges are atrophied and on their palmar surface there are enlarged vessels under the skin. These are at times very irritable, like "chilblains." The nails are becoming smaller.

The Nodules: Little changed since patient was last shown. They vary from about a $\frac{1}{2}$ to 1 cm. in diameter. They are most numerous in the fascial tissues round joints and tendon sheaths, especially on the elbows and backs of the hands. A few are present on the knees and ankles.

Treatment has been carried out with thyroid and electrical baths and the patient considers herself somewhat improved.

Erythema Nodosum, leading to the Detection of Latent Hilus Tuberculosis.

By F. PARKES WEBER, M.D.

THE patient, H. H., aged 10, is a rather fat girl, with slight hypertrichosis of the back of the thorax. Excepting for ear disease, for which a right mastoid operation was performed at the age of seven months, she has usually enjoyed good health. Her enlarged tonsils were excised thirteen months ago. She is the youngest of a family of eight brothers and sisters, who, as well as the mother and father, are all living. The present trouble, which was first noticed on February 21, now (March 3) consists of several large, shiny bluish patches of erythema nodosum in front of the legs, over or close to the tibiæ. There are none on the upper extremities or elsewhere on the lower extremities. No pain has been complained of, and the patient does not now feel ill, though she was ailing for a week before the erythema nodosum attracted attention.

By ordinary examination there is no evidence of disease in the thoracic or abdominal viscera or in the mouth or tonsils, but there is some chronic enlargement of cervical lymphatic glands, and the Pirquet cuti-reaction for tuberculosis is strongly positive (allergic response). Moreover, a Roentgen skiagram of the thorax shows enlarged bronchial glands, doubtless due to tuberculosis. So that there is a condition of bilateral hilus tuberculosis present, as well as possible tuberculosis of cervical lymphatic glands.

In this case the attack of erythema nodosum should, I believe, be interpreted as the manifestation of a mild "tuberculous bacillæmia," due to the "escape" or

[1] See *Proceedings*, 1926, xix (Clin. Sect.), p. 36.

"overflow" of tubercle bacilli from some previously latent tuberculous focus, probably in the lymphatic glands at the roots of the lungs or in the neck. A tuberculous bacillæmia of this nature is one of the causes of erythema nodosum, though there are certainly other infections which may give rise to an attack. It is, therefore, easy to understand why erythema nodosum may occasionally be followed by fatal tuberculous meningitis and generalized miliary tuberculosis.[1]

[1] Cf. F. Parkes Weber, "Erythema Nodosum with Tuberculous Bacillæmia and Meningitis," *Brit. Journ. Child. Dis.*, London, 1924, xxi, p. 119, and "Further Note on Erythema Nodosum," *ibid.*, 1925, xxii. p. 133.

Section of Dermatology.

President—Dr. J. H. Sequeira.

Case of Sarcoid.

By H. W. Barber, M.B.

Mrs. C., aged 57, consulted me (January 21, 1927) about an eruption on the face, of seven years' duration.

Family History.—Father died of "pleurisy," aged 45; mother died of "heart-failure."

Personal History.—Health as a child good; was born on and lived on a farm. When aged 33 had rheumatic fever; at 40 began to have recurrent attacks of erysipelas, accompanied by cervical adenitis. The last attack was seven years ago and the eruption on the face appeared after this.

Condition on Examination.—At first sight the eruption suggests lupus vulgaris, but on the left cheek are three raised bluish nodules, with vessels coursing over them, which are, I think, sarcoid nodules. On both cheeks are scattered patches of flat lupus-like nodules resembling those on the nose and left cheek of the patient in the case of lupus pernio which I have shown on two occasions (*Brit. Journ. Dermat. and Syph.*, vol. xxxvii, No. 3, March, 1925, p. 133, and *Proc. Roy. Soc. Med.*, vol. xx, No. 4, February, 1927, p. 347). These nodules, when tested with a sharpened match, are, perhaps, harder than those of true lupus, but had the large raised nodules not been present I should have diagnosed the case as one of lupus vulgaris.

The hands are bluish, but no nodules can be felt on them.

The physical signs in the heart are suggestive of an old rheumatic myocardial infection, but there is no evident valvular disease.

There are no physical signs indicative of active disease in the lungs.

X-ray Examination (Dr. Lindsay Locke).—Heart shadow, somewhat enlarged to the left. Diaphragm moves evenly and well. Costo-phrenic angles clear. Marked root shadows with many old calcified foci.

The bones of the feet show some increased translucency, but no clear spaces such as may be found in cases of sarcoid.

Von Pirquet Reactions (Dr. Eyre):—

Bovine			*Human*
Per cent.			Per cent.
64 + +	...	...	64 + +
16 ±	...	...	16 +
4 *nil*	...	...	4 ±
			1 *nil*

For the past few weeks the patient has been taking iodine and thyroid internally, and receiving intramuscular injections of sodium morrhuate.

February 2, 1927.—There are symptoms and signs of excessive dosage of thyroid and iodine. Patient has had six injections of sodium morrhuate, and the flat nodules have softened and oozed, forming dry crusts on the surface. The appearances suggest that they are undergoing involution, presumably owing to the effect of the sodium morrhuate.

68 Barber: *Confluent Lichen Nitidus with Lesions on the Tongue*

Confluent Lichen Nitidus with Lesions on the Tongue.

By H. W. Barber, M.B.

Mrs. S. P., aged 59, kindly referred to me by Dr. Semon, whom she consulted in July, 1926, regarding a generalized eruption, which appeared first on the wrists and gradually spread. White streaks on her tongue had appeared at about the same time.

Family History.—Father died, aged 65, of ? "galloping consumption." Mother died aged 95. She has one son and daughter living and well; one child, aged nine months, died from meningitis.

January 31, 1927.—When I first saw the patient the eruption was evidently slowly disappearing. At that time there was an extensive eruption of lichen nitidus of the confluent type with the following distribution:—

Wrists and Forearms.—Confluent patches, evidently fading, on the extensor surfaces of the forearms, particularly the right—infiltrated scaly patches in which the primary papules were in places indistinguishable.

Antecubital Fossæ.—Diamond-shaped patches of close-set papules tending to become confluent.

Neck.—Large area encircling the neck extending up to the hair on the nape, down to the clavicles, and involving the V-shaped area on the manubrium sterni.

Submammary Regions and Abdomen.—Eruption spreads across the mid-line from under both breasts and down to the umbilicus. On the lower abdomen, numerous discrete papules extend out to the flanks and down to the groins.

Back.—Practically free, except between the nates.

Popliteal Spaces.—Closely-set papules as in the antecubital fossæ; a few discrete papules on the inner side of the knees.

Mucous Membranes.—On the tongue are two symmetrical white streaks, and white patches on mucous membrane of the cheeks; formerly white patches on the inner surface of the *lower lip* according to the patient's statement. No sign of the eruption on the vulva.

A biopsy was made on the left forearm, on the outer side of the antecubital fossa. The typical appearances of lichen nitidus are seen in the serial sections submitted.

The great interest of this case is the presence of lesions in the mucous membrane of the mouth, which resemble the appearances of lichen planus in this situation. Dr. Civatte is inclined to consider lichen nitidus as a variety of lichen planus, and has shown me sections from a case in which typical lichen planus and lichen nitidus were both present: in this case there were lesions in the mouth histologically characteristic of lichen planus.

The clinical and histological appearances of lichen nitidus are so definite, and the involvement of the antecubital and popliteal spaces in the confluent type of case so constant, that I feel sure it is a clinical entity distinct from lichen planus. I am satisfied from a careful study of a long series of cases that it is not, as Chatellier suggests, a tuberculide.

Discussion.—Dr. A. M. H. Gray said that this case resembled one described as miliary lichen planus by Dr. Maloney, who differentiated it at some length from lichen nitidus. There was a type of lichen planus in which the differentiation was very difficult. He (Dr. Gray) thought the appearance of the section now shown more like planus than nitidus; the infiltration was flat, whereas in nitidus it was round and deeper in the epidermis than this section showed. Further, there was not, usually in nitidus, that marked thickening of the granular layer. The lesions in the mouth were like lichen planus lesions.

Dr. BARBER (in reply) said that, not knowing the cause, he had no rational treatment to offer, but small doses of iodine seemed to cure the itching and to hasten involution of the lesions.

With regard to the histology, he had collected many serial sections from a number of these cases. Clinically, he regarded the present case as typical lichen nitidus. On looking at the discrete papules—best seen on the lower abdomen—and at the condition in the antecubital fossæ and popliteal spaces, it would be agreed, he thought, that no other diagnosis was possible.

With regard to the microscopical sections, in this case the appearances were not so typical as in other cases he had had of the condition, the reason being that in the section he was now showing there was marked hyperkeratosis over the nodule. Though such hyperkeratosis might occur, as McDonagh had pointed out, it was not usually so definite as in the present case. The thickening of the granular layer referred to was more evident than was usual in lichen nitidus. But even in these sections one saw the apparent eating away of the epidermis by the lichen nitidus infiltration, which was not seen in lichen planus. In lichen planus the epidermis was thickened (acanthosis).

Lupus Erythematosus of the Scalp, associated with Scars of Old Papulo-Necrotic Tuberculide Lesions and of Erythema Induratum on the Arms and Legs.[1]

By H. W. BARBER, M.B.

MRS. C. P., aged 41.

Personal History.—When an infant patient had an operation for enlarged cervical glands; the scar is still present. At the age of 19 she again had enlarged cervical glands, and at this time there were recurring cutaneous lesions on the arms and legs. The lupus erythematosus of the scalp has been present for about four years.

Present Condition.—A well-marked chilblain-circulation, skin of arms and legs being bluish, even when she is at rest in bed; face also dull bluish-red; numerous staphylococcal pustules on the rosaceous area.

On the *forearms*, particularly around the elbows, and extending down the ulnar border, are numerous pitted scars of varying size, suggestive of a former eruption of papulo-necrotic tuberculide.

On the *legs*, around and below the knees, are similar scars, and above the ankles larger ones, suggestive of the deeper subcutaneous lesions of erythema induratum.

On the *scalp* there is an extensive eruption of lupus erythematosus.

Numerous hard discrete glands are palpable in both triangles of the neck.

On admission to hospital the patient had very severe oral sepsis, which has been treated.

Wassermann reaction: Negative.

Complement-fixation Test for Tubercle: Negative.

Von Pirquet Reaction (Dr. Eyre): 64 per cent. + +, 16 per cent. +, 4 per cent. *nil*, 1 per cent., *nil*.

X-ray Examination of the Chest shows calcified nodules at the apex of the right lung and increased fibrosis at the right root.

This case might be cited in favour of the tuberculous origin of lupus erythematosus, but it should be remarked (1) that both lupus erythematosus and the papulo-necrotic tuberculide eruptions occur chiefly in persons with a chilblain-circulation, and their co-existence in the same person, which is not very common, does not necessarily indicate that they are due to the same infection; (2) that in this patient the tuberculide lesions occurred when the tuberculosis of the cervical

[1] This case should be compared with one shown by Dr. Dowling at the last Meeting of the Section.

70 Drake: *Xanthoma;* Dowling: *Epithelioma Adenoides Cysticum*

glands was evidently active—over twenty years ago—whereas the lupus erythematosus appeared only four years ago. The cervical glands, although still palpable, are hard and discrete, and the focus at the apex of the right lung is calcified; (3) that although the von Pirquet reactions are positive, indicating merely that the patient has had tuberculous infection, which is clinically obvious, the complement-fixation test for tubercle is negative.

Xanthoma.

By J. A. DRAKE, M.D.

PATIENT, female, aged 47.

Since August 19, 1926, xanthomatous lesions have been present at inner and outer canthus of both eyes, on the back of the neck and upper part of the chest, that is on exposed surfaces of the skin. Complains of recurrent abdominal pain and nausea, and has had jaundice.

No evidence of diabetes.

Blood cholesterol 338 mgm. per cent., i.e., about twice the normal percentage.

Nothing of importance on abdominal examination.

Dr. F. PARKES WEBER said that he used the term, "sheet-like xanthoma," for large flat xanthomatous areas on the neck and adjacent parts (as in the present case), hardly raised above the level of the normal skin. This sheet-like xanthoma occurred chiefly in cases of very chronic obstructive jaundice, for instance, in so-called hypertrophic biliary cirrhosis ("Hanot's disease"). In Dr. Drake's present case there had, according to the patient, been previously jaundice, and there was probably some chronic hepatic or grave metabolic disease present. The high blood-cholesterol, reported by Dr. Drake, was an important point, and he (Dr. Weber) would like to learn the subsequent course of the case.

Lupus Disseminatus.

By J. A. DRAKE, M.D.

PATIENT, male, aged 23. Underwent operation for enlarged cervical glands when a boy.

Present Condition.—Patches of lupus of various sizes on the face, neck, back and limbs. Very soft and succulent. Diffuse brown infiltration found on diascopy.

Epithelioma Adenoides Cysticum.

By G. B. DOWLING, M.D.

R. F., MALE, aged 22, has on his face a large number of small projecting round tumours, varying in size from a pin's head to a pea. Most of the larger tumours are agglomerated in the naso-genial furrows where they form a confluent mass. In colour the tumours are pearly white, with a faint pinkish tinge; they are translucent. A few of the larger tumours are slightly umbilicated. Occasional milium-like bodies can be seen on the surface of these larger tumours and elsewhere on the face. There is an increased degree of ordinary seborrhœa. The lesions are of five or six years' duration, having first appeared when the patient was about the age of puberty.

Histological section shows bunches and cords of epithelial cells in appearance identical with those of basal-cell carcinoma, usually encircling cavities, some of which contain the remains of a hair. The appearance suggests that the tumour may be arising from the sheaths of the hair follicles or from the sebaceous glands connected with it.

Section of Dermatology 71

Ringworm of the Scalp treated by Thallium Acetate Epilation.

By G. B. DOWLING, M.D.

THE technique advised by Buschke has been followed; that is, a single dose has been given by the mouth in sweetened water. The actual dose given has been in one case 8 mgm. per kilo of body weight, in the other two 8·5 mgm. The first case was treated seven weeks ago; the second and third four weeks ago; the fourth treated was seventeen days ago, and the child is now shedding the hair.

Technique.—The patient should be very carefully weighed, preferably twice on different days. The dose of thallium acetate must be accurately calculated to the nearest mgm. and administered in water. Buschke found that mixing the thallium with food was unsatisfactory, as the epilation partly failed.

The hair begins to loosen on the seventh day, falls out rapidly between the fourteenth and nineteenth day, and is all out by the nineteenth day.

Re-growth begins practically at once. A curious occasional feature in the early stage of re-growth is the presence of enormous numbers of exclamation-mark hairs scattered all over the scalp.

The infected hairs seem to resist epilation more readily than do the normal hairs, but they usually fall a few days later.

Toxic effects have occurred in about 25 per cent. of cases in which I have administered thallium, but as in a number of these cases the treatment is not yet completed the percentage will probably be higher.

All these toxic reactions have been mild, and have been relieved by rest in bed. In practically every case affected, pains in the lower limbs have been complained of, with some loss of appetite in a few cases, and in one case headache was a transient symptom occurring at the time of epilation. The pains in the limbs occur about ten days after the dose, and pass off at about the time of epilation, eight to ten days later. No symptoms of any kind have been observed in the first week.

In Buschke's cases there were no toxic symptoms of any kind, and he was able to produce complete epilation with 8 mgm. per kilo. We conclude therefore that: (1) the thallium used should be biologically standardized; (2) it should be freshly made and absolutely pure; (3) the patients should, if possible, be kept in bed throughout the treatment.

Discussion.—Dr. S. MONCKTON COPEMAN, F.R.S., said that the cases in which he had been using thallium acetate were not comparable with those described by Dr. Dowling. Dr. Semon had that day seen cases treated with this drug in the wards of one of the metropolitan infirmaries, in which he (the speaker) was carrying on this work in conjunction with Professor Dixon, of Cambridge. The patients were almost all old people (one was a woman, aged 78), the subjects of far-advanced cancer, and so nearly moribund that to attempt treatment seemed almost absurd. More thallium was given to these patients than in the dose given to children and young adults, solely administered—so he understood from Dr. Semon—for epilation purposes. In his (Dr. Copeman's) first few cancer cases thallium acetate was given by the mouth, and Members of this Section might be interested to learn that in none of these cases did any epilation occur. The dosage was 1 gr. each day for eight, eleven, thirteen, nineteen and twenty-eight days respectively. In all these cases death had occurred before a longer time had elapsed. Although there had been no alopecia, there had been other symptoms, chiefly nervous, such as, for instance, tingling in the legs, feet, toes, arms and fingers. As oral administration appeared to be unsatisfactory the drug was injected intramuscularly into the gluteal region. Somewhat similar, but—with regard to the effect on cancer—better results followed the change of method. Dr. Semon had estimated that in the cases seen that day at least four times the amount of thallium given to children had been given. Of course, in the cancer cases the patients, being adults, were heavier than children, a fact which would tend to

somewhat equalize the dose according to body-weight. In all these cases alopecia had developed, and in one or two nervous symptoms had appeared. One patient had distinctly mental symptoms and at the end of a week, during which he had had three injections of the drug, he had suddenly manifested delusions of persecution, and it had been necessary to send him to an institution for mental cases. He was now back, and well. In his case there had not been any alopecia at all, though the dosage had been considerably higher than the usual one for children. One patient had had diarrhœa and abdominal pain, and in that case it was considered wise to stop the drug. A week after the administration was stopped the hair began to fall out. That was about a month ago. All the patients were now beginning to recover their hair.

The reason why thallium salts were given in the cancer cases was because of their special action on epidermal tissues, and also because in the periodic scale thallium was placed near to lead, the use of which in cancer had apparently afforded some good results at Liverpool During the last eighteen months many experiments in connexion with the pharmacology of thallium acetate had been carried out on animals in Professor Dixon's laboratory at Cambridge, and workers there found that it had not only an effect on the hair, but also, he understood, on the generative system; evidently it had the same effect as lead in destroying the very early fœtus.

In those animals the effect on the growth of the hair was interesting. When animals were shaved on the abdomen, a comparatively small dose of thallium acetate had the reverse effect, causing the hair to grow more rapidly. When, however, the dose was doubled or trebled, epilation took place as usual.

Obviously certain effects of thallium were very undesirable, and considerable caution should be observed in using it, though its exact toxicity—as compared with that of lead, for instance—had not yet been definitely determined. In order to obtain other effects of the drug, while avoiding epilation, if possible, he (Dr. Copeman) was now using intramuscular injections of a colloidal preparation of thallium iodide, prepared under Professor Dixon's supervision, in the Cambridge University chemical laboratory. This preparation, in contrast to thallium acetate, was extremely insoluble, the idea being that, by securing continuous, very slow absorption, it might perhaps prove possible to avoid, in cancer cases, that special effect which it was sometimes the aim of Members of this Section to attain.

Dr. H. C. SEMON said that in a case he was now treating with krysolgan, in which the same type of symptoms had developed—pain in the joints, and especially at the back of the neck—due probably to the presence of gold in the circulation, he had, two days ago, administered a dose of thiosulphate of soda, in the attempt to antagonize those symptoms. The result was that within three or four hours the patient was completely free from pain, and slept comfortably for the first time since she had been taking the krysolgan. The atomic weights of gold (197·2) and thallium (204) were very approximate (197·2—204), and so for these similar subjective symptoms the thiosulphate of soda might prove to be useful.

Recurring Eruption: ? Artefact.

By G. B. DOWLING, M.D.

THIS girl, aged 13, has had a recurring eruption on the forearms and backs of the hands for two years, and occasionally on the nose. The history is that about two years ago she was associated with a woman who had a bullous eruption, and afterwards "round blisters" developed on the hands and forearms, and sometimes on the nose. The patient is a very neurotic girl, and prone to fits of temper. She began to menstruate at the unusually early age of 10, and she is obviously very over-developed for her age. These spots generally come out when she is particularly irritable, and this is often just before the menses are due. My first diagnosis was neurotic excoriations (type acné excoriée des jeunes filles), because the lesions were round, and she was obviously in the habit of scratching them. But then

I thought they might perhaps have been preceded by a papular urticaria, possibly with vesicles. The second time I saw her she had linear bullæ along the sides of the fingers, as well as the round lesions, the scars of which can be seen now. I believe this is an entirely artificially produced eruption; my diagnosis is not supported, of course, by the mother.

Chronic Relapsing Pemphigus or Dermatitis Herpetiformis in an Old Man with Chronic Lymphocytosis.

By F. PARKES WEBER, M.D.

THE patient, J. G., aged 70¾, German (no Hebrew ancestry), enjoyed good health till four years ago, when the present illness commenced. At first there were only a few isolated bullæ, the formation of which was preceded by local itching and scratching. Since then he has suffered on and off, more or less, from a bullous eruption, with decided exacerbations from time to time. At present, during one of these exacerbations, the bullæ are scattered all over the body and limbs, with the exception of the palms of the hands and the soles of the feet, which have always remained free. The mucous membranes are not affected, though he says that he has occasionally had a few lesions in the mouth. There is no special grouping of the lesions. Local itching mostly precedes the onset of the bullæ, and he says that this itching has sometimes been terrible. According to the patient the bullæ develop on previously normal-looking skin, and this seems to be the case, though there are erythematous spots and patches to be seen, which apparently mark the site of former bullæ.

By ordinary examination of the patient, who is not emaciated and mentally is apparently healthy, nothing abnormal has been found in the thoracic and abdominal viscera, the urine, the eyes, and the nervous system. The spleen and liver are not enlarged, but, owing to the chronic cutaneous trouble, there is moderate enlargement of the superficial lymph-glands (cervical, axillary, inguinal and supracondylar). There is no fever. The gastric contents after a test-breakfast (February 8, 1927) showed no free hydrochloric acid, but the examination was not complete. Brachial blood-pressure; systolic, 120 mm. Hg; diastolic, 65 mm. Hg. The Wassermann reaction is uncertain, owing to "Eigenhemmung." (The blood-serum is not clear, even during the fasting state.) The blood-sugar (fasting) is 0·100 per cent.

The fresh cutaneous bullæ contain very few cells, but most of the cells are eosinophils. A recent blood-count gives 4,288,000 red cells and 37,600 white cells to the c.mm. of blood; a differential count of white cells, from blood obtained with a syringe from a vein, gives: polymorphonuclear neutrophils, 26·3 per cent.; lymphocytes, 68·0 per cent.; monocytes, 2·0 per cent.; basophils, 0·7 per cent.; eosinophils, 3·0 per cent.

This marked lymphocytosis is chronic, for a differential count taken by Dr. F. E. Loewy on August 16, 1925, gave: polymorphonuclear neutrophils, 22·0 per cent.; lymphocytes, 68·6 per cent.; monocytes, 4·7 per cent.; no basophils; eosinophils, 4·7 per cent. The chronic lymphocytosis in this case is very interesting, and it may be compared to that found by Sequeira and Panton in cases of what they termed "lymphoblastic erythrodermia" (*Brit. Journ. Derm. and Syph.*, London, 1921, xxxiii, 391-400).

Another elderly man, L. J., aged 69, who for several years on and off has suffered from similar chronic recurrent pemphigus or dermatitis herpetiformis, has occasionally been an in-patient at the German Hospital, and I have just heard (through the kindness of my colleague, Dr. E. Schwarz) that he is at present quite free from bullæ.

74 Weber: *Persistent Erythema;* MacCormac: *Ringworm of Scalp*

Dr. A. M. H. GRAY said he thought this case belonged rather to the "dermatitis herpetiformis" group, as at present there were erythematous lesions on the body, apart from bullæ. He agreed with Dr. Graham Little, however, as to the practical impossibility of differentiating these cases; and this view was strongly held in America. Some authorities said that the conditions could not be distinguished until the patient had died.

With regard to analysis of the stomach contents, the mere determination of absence of hydrochloric acid, without employing a fractional test-meal, was not of much value unless a fractional test-meal had been given. He asked whether the total chlorides in this case were known. Dr. Bolton had clearly shown that the acidity of the stomach depended largely on the amount of regurgitation from the duodenum.

Postscript (March 28, 1927): The patient, J. G., suddenly developed severe facial erysipelas on March 18, which spread rapidly over the body, and terminated fatally on March 20. He had recovered from an attack of erysipelas about two years previously. A post-mortem examination showed, by microscopic examination, extensive non-inflammatory, systematized, interacinous, lymphocytic infiltration of the liver, characteristic of chronic lymphatic leukæmia. The spleen was about six times the size and weight of a normal spleen, and microscopical examination showed that this was due largely to lymphocytic infiltration, and not merely to the acute fatal erysipelas. There was old, chronic thickening of the splenic capsule. The kidneys, by microscopic examination, showed scattered patches of lymphocytic infiltration. The chronic lymphocytosis and enlargement of the superficial lymph-glands observed during life were, therefore, certainly due to a condition of chronic lymphatic leukæmia. In fact, the supposed "lymphocytosis" was leukæmic and not a true lymphocytosis.—F. P. W.

Persistent Erythema, with Ischæmic Circulation in the Left Foot, possibly in part connected with Prolonged Use of a Plaster Bandage for Tuberculous Disease of the Left Knee.

By F. PARKES WEBER, M.D.

THE case has been described in the *Proceedings* of the Clinical Section Meeting of February 11, 1927.[1]

Dr. H. C. SEMON said he had been called in to treat a case in which peri-arterial sympathectomy had been performed for incipient gangrene, in an elderly man. His foot had been saved by the operation, but subsequently an intractable ulcer had developed in the region of the heel. He (Dr. Semon) thought that this had occurred as a result of the operation, and that care was necessary in deciding as to surgical treatment in these cases.

Ringworm of the Scalp in an Adult.

By H. MACCORMAC, C.B.E., M.D.

THE patient is a railway employee, aged 29. His occupation does not bring him into contact with horses, and it has not been possible to discover the source of his infection. The lesion, which is on the left side of the head, is stated to have developed abruptly four of five weeks ago, and now presents the appearances of a modified inflammatory ringworm of the scalp; the diagnosis has been confirmed by the discovery of a large-spored fungus in the hair.

[1] See *Proceedings*, 1927, xx (Clin. Sect.), 46.

Section of Dermatology 75

Multiple Familial Telangiectases.

By S. E. DORE, M.D.

PATIENT, female, aged 56, for the past fourteen years, has had multiple telangiectases, some of them large macules, with hyperkeratosis of their surface, on the face. She also had them on the tongue and lips, a few on the body, one or two on the hands, and one under a nail. She suffered frequently from nose-bleeding, a symptom noted in other cases of the kind. Her mother suffered from multiple telangiectases, but had, apparently, only a few lesions, which were larger in size. The patient does not know whether other members of the family have been similarly affected. She was treated by electrolysis many years ago, but she faints so readily that the electrolysis had to be stopped. I propose trying CO_2 snow. This is the third case of the kind I have seen. One of the patients (a man) said that the condition had been known in his family for a hundred years. The third patient was a young woman, and no other members of her family appeared to be affected.

Dr. F. PARKES WEBER said that, many years ago, he had published a familial group of the kind.[1] One of the great points of interest was that though the tendency was inborn, the lesions of the skin and mucous membranes manifested themselves or were often first observed at relatively late periods, as in the present case. The nose-bleeding, however, was often noted earlier. The present patient said she had sometimes had slight epistaxis when she was a girl at school; it was a question whether that indicated the time of development of the telangiectatic lesions of the nasal mucous membrane.

Papular Swellings on Eyelids and Forehead: ? Colloid Milium.

By J. H. T. DAVIES, M.D.

THIS patient, a lady, aged 45, has had swellings in her eyelids and the skin of her forehead for ten years. They have been multiplying more rapidly lately. She has been and is well otherwise. She states that her father had a "touch of the same thing." She thinks the swellings vary from day to day,

The eruption consists of 150 or so of papules varying up to the size of a hemp seed forming visible elevations on the skin, but so soft as to be scarcely palpable. When the skin is put on the stretch they appear as round or lobulated, opaque white granules lying fairly superficially in the skin. I have made the diagnosis of colloid milium. I have not yet had an opportunity of cutting sections.

Dr. F. PARKES WEBER said he thought this condition was so-called colloid milium. He had seen it in elderly women, but had never been called upon to treat it.

Mucous Adenomata of Palate: Case for Diagnosis.

By J. E. M. WIGLEY, M.B.

THE patient, a man, aged 37, shows, at the junction of the hard and soft palate, several whitish papules, each having a red, apparently hæmorrhagic, centre. They are quite hard to the touch, and give rise to no sensation. In front of them, immediately behind the incisor teeth, is a red thickened patch, which is slightly

[1] F. Parkes Weber, "Multiple Hereditary Developmental Angiomata (Telangiectases) of the Skin and Mucous Membranes," *Lancet*, 1907 (ii), 160; also "Developmental Telangiectatic Hæmorrhage and so-called Telangiectasia, familial and non-familial," *Brit. Journ. Child. Dis.*, 1924, xxi, 198.

76 Graham Little: *Erythema Scarlatiniforme*

sore. The whole palate is anæmic and the natural folds of the mucous membrane are much exaggerated. The patient has considerable pyorrhœa, probably due to his being a very heavy cigarette smoker. Wassermann reaction negative. He does not wear a dental plate. Dr. Gray has pointed out that the papules very much resemble those in a photograph in a case described by Fordyce and MacKee (*American Archives of Dermatology and Syphilology*, May, 1926). That case had proved to be one of mucous adenomata.

Erythema Scarlatiniforme.

By E. G. Graham Little, M.D.

THE patient came to me this morning with a subsiding eruption and a remarkable history. A year ago a very sudden, universal, scarlatiniform eruption followed after his doing a quite brief clipping of a privet hedge. The inflammation began in the hands and spread very rapidly over the whole surface of the body. It was followed by universal exfoliation and loss of hair and nails. There were no other serious symptoms except a slight rise of temperature for a short time. The present attack commenced exactly like that of a year ago but subsided before any large area had been involved. He has frequently cut the same hedge both before and since without having this reaction. There were no intestinal symptoms with either attack, and he feels otherwise fairly well.

Section of Electro-Therapeutics.

President—G. B. BATTEN, M.D.

Cardio-vascular Syphilis—an X-ray Study.

By Professor FRANZ M. GROEDEL (Bad Nauheim).

[PROFESSOR GROEDEL, after having given an interesting historical survey and described the various forms of cardio-vascular syphilis, proceeded to discuss the value of Röntgen rays as a diagnostic agent.[1]]

I will now proceed to demonstrate a large series of Röntgen photographs; first, twenty-three cases of arterio-sclerosis, then fifty-nine cases of true and mixed syphilitic lesions of the circulatory apparatus.

If we ask ourselves the question, after having seen these X-ray photographs, whether, indeed, they show the characteristic symptoms which point to syphilis, and can inform us of the seat of syphilitic lesions, the answer in some cases will be in the affirmative, the other cases in the negative. The pictures have been shown from different points of view. Nearly all of them refer to lesions of the aorta. Most authors maintain that they have observed, where syphilis of the aorta was present, enlargement and irregularity of outline in the ascendens only. I believe that this unanimity may be the result of auto-suggestion, due to the fact that pathologists persistently claim that syphilitic lesions in the aorta are found in the greatest number near the entry. When we remember, what the pictures clearly show, that aneurysms are to be found on almost every part of the thoracic aorta, we may regard this as a sufficient proof of the error of such claims.

Only by comparing a series of non-syphilitic disturbances of the heart with a series of similar disturbances caused by syphilis, can one determine the characteristic symptoms of the condition. These are here briefly set out.

(1) In severe *aortic sclerosis*, the X-ray shadow of the large vessels shows so great an enlargement that one is tempted to speak of a spindle-shaped aneurysm. But it is not advisable to do so, for even anatomists cannot always be sure whether dilatation or a spindle-shaped aneurysm is present. All parts of the aorta are dilated, but the ascendens in particular, in most cases. The arch comes well forward into the lung area. On the photograph its shadow appears remarkably dark, and easily distinguishable from the lighter shadow of the descendens. Occasionally "plaques" of shadow are observed in the aorta. More frequently the margins of the shadows of the vessels appear to be streaky. This is the result of tangential projection of calcified regions.

(2) Severe *dilatation of the aorta* is also occasionally found in syphilitic aortitis, so that we are again tempted to speak of spindle-like aneurysm. Generally the diffuse dilatation is much less than in cases of true arterio-sclerosis. In the first place the three parts (ascendens, arch and descendens) which appear in the shadow of the aorta are less well differentiated. In a case of syphilitic aorta the border line

[1] It is regretted that owing to lack of space only the part of the paper dealing with the X-ray study can be published in this Section's *Proceedings*. The same applies to the Discussion on the paper.

40 Groedel: *Cardio-vascular Syphilis—an X-ray Study*

is frequently indistinguishable. Further, the arch does not protrude into the field of the lungs, as in sclerosis; it maintains an easy arch-like curve. It is correspondingly less clearly defined in intensity from the other parts of the aorta.

(3) *Sac-like aneurysms* are usually the result of syphilitic aortitis. Occasionally they may be of traumatic origin. The characteristic sign of a sac-like aneurysm is a protrusion of shadow from the line of the shadow of the vessels. The difference between the X-ray diagnosis of aneurysms and tumours cannot be minutely discussed here.

(4) Cases of *endocardial disease* of the aortic valves generally show an intensely dilated and hypertrophic ventricle, so that the shadow of the heart is recumbent and cylindrical. In nearly every case the base of the aorta is somewhat enlarged.

(5) These characteristics are not found in *syphilitic insufficiency of the aortic valves*. The vitium is probably slower in development, and the hypertrophy of the ventricle occurs earlier. In most cases not only is the aorta ascendens enlarged, but there is a slight diffuse enlargement of the whole of the visible aorta.

(6) I have not yet been able to come across a case of *syphilitic stenosis of the aortic valves*.

(7) In cases in which *endocardial heart failure* was present before infection by syphilis, syphilitic lesions may often not be visible in an X-ray photograph. I should be disposed to assume that in such cases the aorta is less subject to syphilitic infection, but I find no confirmation of this view in any work on the subject. If treatment of the heart has no result, I conclude that such patients are suffering from syphilitic disease of the cardiac muscle, liver, etc.

(8) In cases in which the *coronary vessels* have been closed by a *syphilitic lesion of the aorta*, the X-ray photograph shows the characteristics of aortic disease, as mentioned above.

(9) In *syphilitic disease of the cardiac muscle* the relatively slight enlargement of the heart is often remarkable.

(10) In *syphilis of the liver* there is often found a secondary myocardial lesion, with no characteristic symptoms shown by X-rays.

(11) My own considerable researches have shown me that *syphilitic lesions of the lungs* are easily recognizable in X-ray photographs. There are present nodular gummata of the lungs, either coarse or fine, frequently accompanied by cirrhotic processes. Or the latter may be found isolated; either with cirrhotic stenosis, or sometimes with pneumonic processes; finally, there may be cirrhotic processes associated with emphysema and asthma. Nevertheless all these symptoms may be, and frequently are, misunderstood, in connexion with heart and lung affections. The best results in such cases are obtained by the use of strong antisyphilitic remedies.

The slides exhibiting diseases of the cardiac muscle and aorta showed that we frequently find shadows in the lungs which would appear to point to true pulmonary syphilis of the lungs; in any case these are not really typical of the X-ray pictures of cardiac stasis of the lungs. In syphilis there is a remarkably frequent occurrence of exudation from the right side of the lungs; in cardiac stasis exudation from the left side preponderates. I am willing to assume that in many cases of circulatory disturbance due to syphilis, not only the heart and liver symptoms, but also the lesions of the lungs, may be due to syphilitic infection.

(12) There is not much to be said on the subject of X-ray photographs of *congenital syphilis*. I suppose that in such cases the same symptoms are present as in acquired syphilis.

Mixed cases may arise—e.g., vitia with syphilitic circulatory disturbance, or the latter with sclerotic lesions of the cardiac muscle or the aorta. In these cases,

differential diagnosis may not be necessary, as the treatment will in any event have to be of a mixed nature.

Lastly, exact diagnosis is of extreme importance, since the methods of treatment for syphilitic and non-syphilitic disturbances of the heart are quite different. Once we have seen a large number of cases which have undergone heart treatment for years without benefit, improving almost in a single night with the introduction of specific treatment, we feel that the trouble taken in establishing the diagnosis has been well worth while. I take a view diametrically opposed to that of most authors, in maintaining that the circulatory apparatus is generally able to stand the treatment. In cases in which the lesions of the vessels and heart show no tendency to heal, or in which the spirilla have become immune to antisyphilitic treatment in any reasonable dose, it may be discovered that treatment has been begun too late.

Discussion.—Dr. JOHN PARKINSON said that in spite of the established pathological teaching that syphilitic aortitis was generally more pronounced in the ascending portion of the arch of the aorta, the lecturer had held that this fact was seldom of diagnostic value in its radiographic diagnosis. Though this might be true generally, he (Dr. Parkinson) had seen sufficient cases in which the first part of the aorta was so disproportionately enlarged, and yet without saccular aneurysm, that the diagnosis of syphilitic aortitis at once occurred to him.

The lecture had been of great value in showing how much could be learned by X-ray examination in a single ætiological variety of circulatory disease—that due to syphilis. The slides shown had amply proved its value in distinguishing atheromatous aortic disease from syphilis, but he (Dr. Parkinson) would draw attention to the fact that the aortic outline might be used in helping to distinguish between hyperpiesis without gross arterial change, and the atheromatous aorta where the blood-pressure might be little if at all raised. Professor Groedel had rightly insisted that combinations of atheroma, syphilis, and high blood-pressure, might produce a complicated picture. If so much could be learned by radioscopy in individual patients suffering from this group of vascular disease (and its value might be no less in the rheumatoid group and in congenital malformations), we could not afford in the interests of these important patients to rely upon percussion so much as in the past. Percussion still had a place in the diagnosis of cardiac disease but it was rapidly becoming inadequate and this could not be seen better than in the instance of cardio-vascular syphilis, as had been so ably demonstrated by Professor Groedel.

Dr. STOLKIND said he had come to the conclusion that as a rule, *where the X-ray showed dilatation of the aorta* in adults, then *syphilitic aortitis was present.*

Dr. JAMES F. BRAILSFORD (Birmingham) showed a series of lantern slides of a case of aneurysm of the abdominal aorta, which demonstrated a further radiographic appearance of great value in the diagnosis of this lesion, i.e., scalloping of the bodies of the lumbar vertebræ, due to erosion of the bone by the aneurysm except near the intervertebral discs, which were not eroded, and projected into the aneurysmal sac. This being so, the vertebral bodies did not "fall together" as in tubercle or new growth, and an antero-posterior radiograph might either reveal no abnormality or, as in this case, an appearance which had been on several occasions diagnosed as osteo-arthritis. In view of the statement made that aneurysm and its diagnosis was of no more interest to the physician than a fungating breast tumour to the surgeon, he (Dr. Brailsford) would say that this patient had had severe symptoms for two years for which he had sought treatment at various hospitals. The condition had been consecutively diagnosed and treated as rheumatism, lumbago, osteo-arthritis and gastro-intestinal disturbance. He eventually came to the Queen's Hospital, Birmingham, with the diagnosis of "twisted kidney," but after examination the patient was sent to the X-ray department for a barium meal examination with the provisional diagnosis of "carcinoma of the splenic flexure." It was on the appearance of the lateral radiograph that the diagnosis of "aneurysm of the abdominal aorta" was made. A post-mortem two months afterwards confirmed the diagnosis.

Dr. GEORGE VILVANDRÉ said that the case of aneurysm of the abdominal aorta shown by the last speaker (Dr. Brailsford) was similar to one known to him (Dr. Vilvandré),

42 Groedel: *Cardio-vascular Syphilis—an X-ray Study*

but the X-ray diagnosis of aneurysm was not always as readily made, for only a very few cases exhibited erosion of the vertebræ. In his own case, which had also been seen in Vienna and Paris, the diagnosis was not definitely made from echinococcal cyst for some time, but was settled by erosion of the vertebræ appearing. Remarks had been made manifesting little interest in aneurysm, but the diagnosis of this complaint had to be made, and the necessity for doing this often devolved upon the radiologist. Though frequently decisive the Wassermann test was not always conclusive; it might be slightly positive in the hands of an expert and negative in the hands of another when made on the same patient at the same time. The radiological evidence became, then, even more necessary. The field of X-ray diagnosis in cardio-vascular disease was becoming more important, and with the electrocardiograph encroaching upon and even in some cases superseding the stethoscope, all facilities should be given to the radiologist to enable him to acquire more knowledge in that branch of medicine. This could only be done by the assemblage of correlative facts, clinical and radiological, and ultimate diagnosis (post-mortem). A member of the X-ray department of a hospital might wisely be set apart for such a purpose, working entirely with the physician for heart disease.

Dr. S. W. PATTERSON said that syphilitic myocarditis showed on the X-ray screen a large flabby heart flattened out on the diaphragm, with weak beats, often slow and accompanied by a low blood-pressure.

Section of Epidemiology and State Medicine.

President—Dr. S. Monckton Copeman, F.R.C.P., F.R.S.

The Incidence of Pneumonia.

By W. St.C. McClure, M.R.C.S., L.R.C.P., D.P.H.

(Deputy Medical Officer of Health, Manchester.)

More than 70,000 deaths occur annually in England and Wales from pneumonia and bronchitis. The relative number of deaths ascribed to lobar pneumonia, broncho-pneumonia and bronchitis varies according to the fashion, or custom, in regard to the form of certification in vogue from time to time. It is consequently impossible to compare, with complete accuracy, the number of deaths over a period of years from any one of these diseases.

In England and Wales during the last thirty years the pneumonia and bronchitis mortality curves follow each other very closely, and it would appear that some measure of stability in the form of certification has been attained (Table I). In

Table I.—Death-rates per million from Pneumonia, all Forms, and Bronchitis, England and Wales, 1897-1925.

Year	Pneumonia	Bronchitis	Total
1897-1900	1,216	1,256	2,472
1901-1905	1,274	1,244	2,518
1906-1910	1,234	1,094	2,328
1911	1,040	1,013	2,053
1912	1,024	1,089	2,113
1913	1,025	1,071	2,096
1914	1,084	1,087	2,171
1915	1,356	1,437	2,793
1916	1,065	1,248	2,313
1917	1,141	1,249	2,390
1918	1,654	1,232	2,886
1919	1,058	1,238	2,296
1920	987	1,010	1,997
1921	916	889	1,805
1922	1,073	1,073	2,145
1923	870	852	1,724
1924	1,003	973	1,976
1925	951	906	1,857

Scotland, since 1891, the pneumonia death-rate has become progressively higher, whilst the bronchitis rate has fallen from 165 in the decennium 1891-1900 to 90·8 in the quinquennium 1921-1925, a drop of 45 per cent. This apparent increase of pneumonia mortality in Scotland is probably due largely to the transference of bronchitis deaths. The combined rate from bronchitis and pneumonia has fallen to the extent of 23 per cent., which is comparable with that which has occurred in England and Wales over the same period.

In England and Wales the combined rate has fallen from 247 in the period 1897-

1900 to 190 in the period 1921-1925, a decline of 23 per cent., the respective percentage figures for bronchitis and pneumonia being 21 and 25.

Pneumonia mortality is heavier in the northern than in the southern group of counties, including London. The relative death-rates in these two areas in the period 1911-1914 were 1314 and 903, showing an excess in the north of 31 per cent. Comparable rates in the period 1921-1925 were 1,223 and 840, the relative excess in the north being approximately the same. Whilst the decrease in England and Wales, as a whole, was 9 per cent., in the north and south it was 7 per cent. The relatively high mortality in the north is therefore maintained (Table II).

TABLE II.—DEATH-RATES FROM PNEUMONIA, NORTH, MIDLANDS, AND SOUTH, INCLUDING LONDON, 1921-1925.

Year	North	Midlands	South
1921	1,164	778	798
1922	1,286	900	1,017
1923	1,150	720	720
1924	1,293	837	877
1925	1,222	824	789

The mortality from pneumonia in England and Wales varies from year to year. The north, the midlands and the south all share in any fall or rise which occurs in any one year (Table II). There is apparent, apart from influenzal occurrences, no such periodicity as is found in certain other infections. Therefore, some factor must be influencing the prevalence of the mortality from these diseases which is common to the whole country.

TABLE III.—DEATH-RATES FROM ALL FORMS OF PNEUMONIA AND MEAN ANNUAL TEMPERATURE (DISTRICT VALUES), ENGLAND AND WALES, 1911-1925.

Year	Death-rate	Mean annual temperature
1911	1,216	50·3
1912	1,024	49·2
1913	1,025	49·8
1914	1,084	50·1
1915	1,356	48·5
1916	1,065	48·9
1917	1,141	47·8
1918	1,654	49·5
1919	1,058	47·8
1920	987	49·6
1921	916	51·1
1922	1,073	48·0
1923	870	48·7
1924	1,003	49·0
1925	951	48·9

Climatic conditions doubtless play a part in determining the incidence of catarrhal affections of the respiratory system. The influence of the climate upon health, however, is a subject which has been incompletely studied. Neither extreme heat, nor extreme cold, nor extreme variability, presents the optimum conditions for physical health.

In nine representative cities of America, in which the mean annual temperature ranged from 44·1° F. to 69·1° F. during 1911-1917, there was less pneumonia amongst the white population of the four warmer cities than of the four colder cities. Nevertheless, the warmest city did not have the lowest rate, nor did the coldest city have the highest.

In England and Wales the mortality from pneumonia is affected by atmospheric

temperature. Table III shows the mean annual temperature and the death-rates from all forms of pneumonia over a series of fifteen years. The following table gives the number of deaths from pneumonia in England and Wales during the first quarters of each of the years 1912-1925, and the mean temperature of corresponding periods. If

TABLE IV.—NUMBER OF DEATHS FROM PNEUMONIA IN ENGLAND AND WALES DURING THE FIRST QUARTER OF EACH YEAR, AND THE MEAN TEMPERATURE (GREENWICH) OF CORRESPONDING PERIODS, 1912-1925.

Year	No. of deaths	Mean temperature
1912	13,110	43·6
1913	13,865	42·4
1914	13,599	42·5
1915	18,717	40·8
1916	12,648	41·5
1917	16,284	36·4
1918	14,506	42·3
1919	20,155	38·1
1920	13,814	44·3
1921	12,836	45·5
1922	16,605	41·2
1923	10,517	44·8
1924	16,678	39·8
1925	12,123	42·2

these figures be charted the similarity of the curves will be evident and the relationship of the two in the series of first quarters is so close that, given the mean temperature of the first quarter of any one year, it is possible to calculate, approximately, the pneumonia death-rate for that period. The coefficient of correlation between the number of deaths and the mean temperatures during the first quarters of the years 1912-1924 works out at $-0{\cdot}768 \pm 0{\cdot}076$.

The influence of air temperature is not so apparent when the relative means of different areas are considered together with the respective death-rates. The mean annual temperature in the south is consistently about two degrees higher than that in the north, whereas the mean temperature of the northern area varies but slightly from that in the midlands (Table V). It might therefore be expected that, if air

TABLE V.—MEAN ANNUAL TEMPERATURE IN THE NORTH, MIDLANDS AND SOUTH OF ENGLAND AND WALES, 1911-1925.

Year	N.W. and N.E. (Average)	Midlands	S.W. and S.E. (Average)
1911	49·2	49·6	50·8
1912	48·0	48·2	49·8
1913	48·8	49·0	50·5
1914	49·3	49·6	50·5
1915	47·4	47·6	49·1
1916	48·2	48·0	49·6
1917	47·1	47·3	48·2
1918	48·7	49·1	50·1
1919	46·7	47·2	48·5
1920	49·5	49·0	50·4
1921	50·3	50·8	52·0
1922	47·3	47·5	48·8
1223	47·9	48·2	49·6
1924	48·3	48·5	49·7
1925	48·1	48·5	49·7

temperature is the all-important factor, pneumonia incidence in the north and midlands would be similar, and that in the south it would be relatively low. Such is not the case. The ratio between the pneumonia death-rates in the north and midlands,

taking the average of two periods, 1911-1914 and 1921-1925, is 1·42 to 0·1, whilst the ratio between death-rates in the midlands and south, including London, is 1·02 to 0·1 (Table VI).

TABLE VI.—AVERAGE DEATH-RATES FROM PNEUMONIA IN THE NORTHERN, MIDLAND AND SOUTHERN COUNTIES OF ENGLAND AND WALES, 1911-1914 AND 1921-25.

Area	Period	Average population	Average number of deaths	Average death-rate
North	1911-14	12,287,195	16,151	1,314
	1921-25	12,932,780	15,817	1,223
Midland	1911-14	11,472,851	9,795	968
	1921-25	12,139,827	9,859	812
South	1911-14	10,398,633	9,494	903
	1921-25	10,508,909	8,829	840

In Manchester, a typical northern town, the coefficient of correlation for the years 1925 and 1926 between the weekly mean temperatures and the weekly number of notified cases of pneumonia having their onset in those several weeks is 0·703 ± 0·047. An attempt to correlate the number of cases occurring daily, according to the date of onset of illness, with the daily temperature showed no definite correlation. It might be thought that fluctuations of temperature, rather than the annual means, should show some definite relation to pneumonia mortality. An effort to measure the relation between the extremes of temperature experienced in successive weeks in Manchester, and the respective number of pneumonia cases occurring in those weeks, or in the weeks following, produced negative results. This may be due to faulty data regarding the dates of onset of illness, or, if the data are correct, it may indicate that the comparatively small fluctuations of temperature which are usual in this country have no serious effect upon pneumonia incidence.

All these considerations contribute in showing that, although there is some direct relation between air temperature and pneumonia mortality, there are other factors of predominating importance which are responsible for the greater incidence of pneumonia in the northern compared with the southern counties.

It is in the large towns that pneumonia prevalence is excessive, and it is the environmental conditions of these towns which are the determining cause. Fogs of a type peculiar to large towns are harmful. In order to demonstrate the effect of black smoke on health in connection with fogs, particulars were collected in Manchester regarding the deaths from pneumonia in each of the six weeks preceding, and in each of six weeks containing and following fifty-nine consecutive fogs in the years 1897 to 1910. Only fogs of two or more days' duration were used. The results are shown in Table VII. The figures express a probability that the increase in the number of

TABLE VII.—PNEUMONIA AND FOGS, MANCHESTER, 1897-1910.

	Weeks preceding the fog.						Weeks containing and following fog.					
	6	5	4	3	2	1						
Number of deaths from pneumonia	1351	1389	1345	1442	1442	1434	1573	1638	1657	1710	1631	1589
Number of deaths from bronchitis	1317	1301	1330	1526	1479	1627	1808	1864	1848	1699	1729	1749

deaths was partly due to fog or to some accompanying phenomenon, fog merely accentuating normal conditions. In a recently made investigation,[1] Russell concludes that although the number of respiratory deaths in the week following fog is more highly correlated with the fogginess than with the temperature of the previous

[1] *Lancet*, 1926 (ii), 1128.

week over certain periods, yet, on the average, a low temperature exercises a greater influence.

The direct influence of fog on the general trend of the pneumonia death-rate is not great. Recorded fogs are fewer now in Manchester than in the past, but the heavy decline in the number of fogs has not been associated with any decline of corresponding magnitude in the number of deaths from pneumonia and bronchitis, as shown by the following figures for Manchester:—

Period	Number of recorded fogs	Death-rate from pneumonia and bronchitis
1891-1900	416	247
1901-1910	234	242
1911-1920	107	258
1921-1925 (5 years)	57	288

Nor is any close association observed if individual years be examined. Table VIII gives the number of fogs and the death-rates in Manchester for pneumonia and

TABLE VIII.—YEARLY NUMBER OF RECORDED FOGS AND THE CORRESPONDING DEATH-RATES FROM PNEUMONIA AND BRONCHITIS, MANCHESTER, 1916-1925.

Year	Days of fog	Death-rate Pneumonia	Death-rate Bronchitis	Total Death-rate
1916	15	125	160	285
1917	19	122	144	266
1918	5	184	137	321
1919	14	124	164	288
1920	10	117	141	258
1921	5	124	143	267
1922	7	137	171	308
1923	7	130	145	275
1924	12	133	163	296
1925	26	127	181	308

bronchitis in each of the years 1916 to 1925. There appears to be no correlation. For instance, in 1916 the number of fogs was fifteen and the death-rate 285. In 1922 fogs numbered seven and the rate was 308. In 1925 there were twenty-six recorded fogs and the death-rate was again 308.

Although, therefore, fog may exert some influence upon pneumonia prevalence, such influence is, in the mass, unnoticeable and represents only a minor and unimportant factor in the epidemiology of these diseases. Fog is only a contributor to those conditions which tend to occlude the rays of the sun. Light is shut out from the northern cities of England over a great part of the year. The average annual percentage of possible sunshine during the period 1881 to 1915 in certain towns was as follows: Manchester 24, Hull 22, Southport 35, Blackpool 36. In the northern area Manchester is above that of Hull only.

Records of light tests made in Manchester and Timperley, a district within seven miles of the centre of the town on its southern border, show clearly the effect of town conditions upon the amount of light and upon the quantity of ultra-violet rays which are able to penetrate the prevailing gloom (Table IX). The light tests

TABLE IX.—PERCENTAGE OF LIGHT RECEIVED IN MANCHESTER AS COMPARED WITH TIMPERLEY (100).

Year	Jan.	Feb.	March	April	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.
1925	38	43·5	91	73	57	53	68·5	60	64·5	55·5	47	39
1926	43	52	55	51	50	49	66	61	48·5	64·5	50·5	37

were made by the iodine method, and the tests for ultra-violet rays by the acetone blue method. It will be noted that throughout the year Manchester receives but 54 per cent. of the light received in its more favourably situated suburb, and that during the winter months the percentage is even lower. As regards the ultra-violet rays, records made during the last nine months show that, as compared with Timperley, 36 per cent. of the rays are, on the average, occluded.

54 McClure: *The Incidence of Pneumonia*

In the words of Leonard Hill, smoke pollution cuts off from the cities ultra-violet rays, warmth and light, destroys green food, the primary source of vitamins, making the streets and parks filthy and dismal, and driving the people away from health-giving outdoor exercise into unhealthy, sedentary and indoor amusements.

Pneumonia mortality is greater in county boroughs than in urban and rural districts. This statement holds good, whether speaking relatively of districts in the north or in the south.

Pneumonia is an infectious disease, and is spread from person to person, and precedent catarrhs are rapidly disseminated amongst persons living in close contact with one another. Like other infections, the spread is favoured by the aggregation of people.

Measles, whooping-cough, scarlet fever and, to a less extent, diphtheria, are more prevalent in the north than in the south, and more so in towns than in rural districts.

TABLE X.—DEATH-RATES PER 100,000 FROM CERTAIN INFECTIOUS DISEASES IN THE NORTHERN AND SOUTHERN COUNTIES (INCLUDING LONDON) AND IN THE COUNTY BOROUGHS AND RURAL DISTRICTS OF ENGLAND AND WALES, 1922-1925.

	Scarlet fever	Diphtheria	Measles	Whooping-cough	Pneumonia
North	3·8	6·2	19·5	15·8	123·4
South	2·4	9·9	11·2	11·4	84·8
Percentage excess in North	37	−37	42	23	31
County Boroughs	3·4	7·8	18·8	16·2	121·4
Rural Districts...	1·7	4·3	6·4	10·2	65·8
Percentage excess in County Boroughs ...	50	46	66	37	46

Table X gives the figures for north and south, including London, and for the county boroughs and rural districts during the years 1922-1925. If London be excluded, the relative excess in the north is greater and diphtheria falls to some extent into line. It will be seen that the excess of mortality from these diseases is relatively as great as, or greater than, is the case with pneumonia. There is no evidence that this undue prevalence is due directly to fogs or lack of sunshine. Nor is it due to poverty, for those persons living in the poorer areas are apparently less susceptible to these diseases. Although climatic and atmospheric conditions may contribute to the prevalence of pneumonia by lowering the vitality of the individual, their influence is neither predominant, nor even prominent.

Distribution of population is the important factor. The greater the density of population in any area, the higher is the mortality from pneumonia.

The occurrence of a higher pneumonia mortality in towns than in rural districts is not peculiar to this country. Statistics of the registration states of America indicate that deaths from pneumonia in the cities exceed those in the rural districts by about 50 per 100,000, the rates being higher in the cities at all ages (Table XI).

TABLE XI.—DEATH-RATES FROM PNEUMONIA IN THE CITIES AND RURAL DISTRICTS OF THE REGISTRATION STATES OF AMERICA, 1911-1917.

	1911	1912	1913	1914	1915	1916	1917
Cities ...	153	152	156	149	156	165	178
Rural ...	105	103	102	97	106	112	121

There is considerable evidence to show that the excess of deaths from pneumonia in the northern counties of England and Wales is due to the greater aggregation and density of the population compared with the south. The population of the northern counties in 1925 was about 13,071,890, and of the southern counties it was 6,140,970, or, including London, 10,752,970. In the north 55 per cent., or more than half of the people, live in towns with populations of over 50,000, whereas 25 per cent. are in communities of less than 20,000. In the counties of the southern group the position is reversed, 25 per cent. of the population live in towns of over 50,000

persons, and 48 per cent. in communities of less than 20,000. If London be included, adding an urban population of 4,612,000, the advantage still remains with the south (Table XII).

TABLE XII.—DISTRIBUTION OF POPULATION IN NORTH AND SOUTH, 1925 (EXCLUDING LONDON).

	North			South		
In communities of:—	Urban	Rural	Total	Urban	Rural	Total
Under 10,000 ...	1,063,280	372,070	1,435,350	684,201	473,180	1,157,381
10 to 20,000 ...	1,458,050	489,060	1,947,110	671,350	1,097,556	1,768,906
20 to 50,000 ...	1,844,040	627,900	2,471,940	1,186,790	469,659	1,656,449
50 to 100,000 ...	1,456,130	292,660	1,748,790	596,484	—	596,484
100,000 and over ...	5,468,700	—	5,468,700	961,750	—	961,750
Total ...	11,290,200	1,781,690	13,071,890	4,100,575	2,040,395	6,140,970

Pneumonia mortality, both in the northern and southern counties, varies directly with the degree of aggregation of the several populations. The northern counties, placed in order of their pneumonia mortality-rates reckoned over the last three years, counties with the higher rates being placed first, are Lancashire, Durham, Yorkshire, Northumberland, Cheshire, Cumberland, Westmorland. This is the order in which the counties are placed if they be classified according to the percentage of the population of each living in communities of 50,000 or over, except that Yorkshire and Durham change places (Table XIII). And it will be noted that if the counties

TABLE XIII.—DEATH-RATES FROM PNEUMONIA AND DISTRIBUTION OF POPULATION IN THE NORTHERN COUNTIES, 1923-1925.

County	Pneumonia death-rate	Per cent. of population living in communities of over 50,000	No. of persons per acre. (Census 1921)
Lancashire	132	65	4·11
Durham	129	53	2·27
Yorkshire	126	55	1·05
Northumberland	111	50	0·57
Cheshire	91	35	1·50
Cumberland	91	30	0·28
Westmorland	60	0	0·13

be grouped in the order of the density of their population, those with the larger number of persons per acre have a higher death-rate from pneumonia than those with the smaller number of persons per acre.

The same relation between the mortality-rate and crowding on area exists in the several towns of Lancashire with individual exceptions. There are in Lancashire 15 county boroughs, in 8 of which the number of persons per acre exceeds 20, and in 7 of which the number per acre is less than 20. Taken in these two groups the average pneumonia death-rate for the three years 1923-1925 was, in the first group 146, and in the second group 110. Further, it can be shown that in a representative large town a similar relationship is evident in the several districts of that town. If the 35 wards of Manchester be grouped into those in which the number of persons per acre is below 50, between 50 and 110 and 100 or more, the pneumonia mortality becomes progressively greater in each group. This is so both for lobar and for broncho-pneumonia, but the proportion of cases of the latter disease is relatively much greater in the more crowded areas than is the case with lobar pneumonia (Table XIV).

TABLE XIV.—DEATH-RATES PER 100,000 FROM LOBAR AND BRONCHO-PNEUMONIA IN GROUPS OF WARDS, MANCHESTER, 1925-1926.

Wards	Population	Average death-rate		Total Death-rate
		Lobar	Lobular	
With less than 50 persons per acre ...	342,135	29	33	62
With 50 to 100 persons per acre	291,716	44	69	113
With 100 or more persons per acre ...	124,354	59	77	136

Other data, also, go to show that overcrowding on area influences the broncho-pneumonia rate much more than it does the lobar pneumonia rate. This may indicate that broncho-pneumonia is more infectious, and more easily spread from person to person, than the lobar variety. The relationship between overcrowding on area and pneumonia mortality in the various wards of the city is true in the gross, but there are exceptions, the most outstanding of which is given by two wards: (*A*) New Cross, and (*B*) Beswick. Ward (*A*) has a population of 29,713, and Ward (*B*) of 32,611. The wards are contiguous and have a similar type of population. The number of persons per acre in Ward (*A*) is 98, and in Ward (*B*) 128, yet the mortality rate in (*A*) is 196 and in (*B*) 105. It is possible that this difference is due, in part, to the greater overcrowding in individual houses in Ward (*A*). The figures in Table XV are suggestive in this respect, and there can be no doubt from a study of Table XVI that overcrowding in individual houses is a prominent factor

TABLE XV.—ACCOMMODATION PER PERSON IN THE WARDS OF NEW CROSS AND BESWICK (INVESTIGATED CASES OF INFECTIOUS DISEASE), 1925 AND 1926.

	New Cross		Beswick	
No. of rooms per person	No. of persons living in infected dwellings	Per cent. of total	No. of persons	Per cent.
Under 0·3 ...	138	9·3	22	1·6
0·3 to 0·5 ...	303	20·6	210	15·7
0·5 to 0·7 ...	553	37·6	490	36·6
0·7 to 1·0 ...	185	12·5	271	20·3
1·0 and over ...	292	19·0	345	25·8

in the spread of these diseases. It is noticeable that here again lobar pneumonia is influenced to a much less extent than broncho-pneumonia by immediate environmental conditions.

TABLE XVI.—ACCOMMODATION PER INDIVIDUAL OF PERSONS LIVING IN INFECTED HOUSES. PNEUMONIA, 1925-1926.

Accommodation per individual	Number of persons living in infected dwellings	Per cent. of total number of persons living in infected dwellings	Percentage distribution of population, Census 1921
Lobar.			
Under 0·3 ...	161	2·5	0·5
0·3 to 0·5 ...	816	12·8	7·4
0·5 to 0·7 ...	1,935	30·4	24·2
0·7 to 1·0 ...	1,258	19·8	19·0
1·0 and over ...	2,191	34·4	49·0
Lobular.			
Under 0·3 ...	343	5·3	0·5
0·3 to 0·5 ...	1,368	21·3	7·4
0·5 to 0·7 ...	2,335	36·4	24·2
0·7 to 1·0 ...	937	14·6	19·0
1·0 and over ...	1,424	22·2	49·0
Influenzal.			
Under 0·3 ...	12	0·6	0·5
0·3 to 0·5 ...	201	9·4	7·4
0·5 to 0·7 ...	640	30·0	24·2
0·7 to 1·0 ...	383	17·9	19·0
1·0 and over ...	895	42·0	49·0

From a study of the age incidence of these two varieties of pneumonia, from the difference in the class of persons attacked and from the different distribution of cases over the year, it is clear that the two diseases under consideration are distinct entities from an epidemiological point of view. All the figures which have been quoted tend to show that overcrowding on area, and in individual houses, is the

essential contributing factor in the spread of pneumonia. In the case of lobar pneumonia the immediate environmental conditions are of less importance. It is possible that the incidence of lobar pneumonia depends more upon the incidence of human carriers of the pneumococcus, which varies from time to time, but becomes greater during the winter months immediately preceding the period of the year when mortality from pneumonia is greatest.

Conclusions.

(1) Pneumonia is endemic in this country. The excess of pneumonia in large towns is not due to differences of age distribution, the mortality-rates in towns being higher at all ages.

(2) Fog and black smoke affect contiguous districts in our cities equally, but it is only in overcrowded districts that the mortality is excessive.

(3) Climatic conditions are contributing factors. It is not the cold weather in itself which causes pneumonia,[5] but the behaviour of persons in cold weather which induces colds and catarrhs. It is not possible to draw a distinct line between pharyngitis, laryngitis, bronchitis and broncho-pneumonia. The majority of the population suffer from one or more colds during the year. After an investigation in Pittsburgh, U.S.A., it was estimated that the number of common colds per person each year was three, and, out of 2,507 cases of pneumonia, it was found that 71 per cent. had a history of preceding cold.

An endeavour was made in Manchester to ascertain the origin of 1,000 cases of pneumonia in children under five years of age during 1925 and 1926. 517, or 51·7 per cent. of the children had previously suffered from pneumonia or bronchitis, or were the subjects of repeated colds. The percentage in the case of lobar pneumonia was 48, and in broncho-pneumonia 56. It would appear, therefore, that pneumonia, especially broncho-pneumonia, frequently has its origin in a common cold or attacks those whose vitality is lowered by chronic catarrhal conditions. This catarrhal state is most common amongst children living in an overcrowded environment, in badly ventilated living and sleeping rooms, faced as they are by the chances of mass infection repeated over and over again.

(4) There is, therefore, considerable evidence showing that overcrowding on area, and in individual houses, is directly responsible for the high incidence of pneumonia. If this view be accepted, the remedy lies in the development of our housing and town-planning schemes. Every additional open space provided in our towns, every slum area demolished, whether large or small, every effort made to clear the centre of our cities and to re-house under hygienic conditions will tend to reduce the mortality from these diseases.

I have to thank the Medical Officer of Health for the City of Manchester for permission to carry out this investigation.

References.

[1] *Registrar General's Annual Supplements.* [2] *Annual Reports, Scottish Board of Health.* [3] "Book of Normals." [4] Vaughan, "Epidemiology and Public Health." [5] *Annual Reports Medical Officer of Health, Manchester.*

Discussion.—Sir William Hamer: This very interesting paper calls to mind the finding of Chalmers that epidemic diseases of the central nervous system are *endemic* in Glasgow. Dr. McClure holds that pneumonia, too, is an endemic disease and has a periodicity identical with that of influenza. In the fifth section of the "Medical Observations," however, influenza appears in a complete setting of cerebro-spinal, pulmonary and gastro-intestinal prevalences. Thus Sydenham, describing the "Constitution" of 1675, says, "The fever exhibited the

symptoms of dysentery and diarrhœa . . . it attacked the head . . . but everyone who had any practice among the sick knew the predominance of the fever and knew that the aforesaid dysentery and diarrhœa were symptoms rather than essential and primary diseases." Then came "coughs and catarrhs . . . the fever, however, remained the same as before." He adds, "All that I know is what I know well . . . that all the diseases which have originated from it have been the same." There follow detailed descriptions of the precursory "lethargies" and "stupors," of the widespread July prevalence, of the pneumonias of October and November and of the return of the "stupors" and "lethargies." The voice speaks from the far-away seventeenth century, but how accurately the description fits the 1918 constitution! It is not merely pneumonia, then, it is not merely cerebro-spinal fever that concerns us, our quarry is that elusive endemic disease "influenza in mufti." Some of those present to-night may live to see the day when prevalent obsessions regarding clinical entities, pathognomonic signs and immutable causal organisms will cease to obscure great issues. Honest Dogberrys will then admonish any die-hard Borachios and Conrades who still persist in disgracing the Lady Epidemiology before the whole assembly; will tell them "it is proved already that they are little better than false knaves, and it will go near to be thought so shortly"; and will appropriately intimate to them that they "will be condemned into everlasting redemption for this."

Professor GREENWOOD said he doubted whether the relation between temperature and mortality from pneumonia could be satisfactorily evaluated by means of data covering so wide an area as those of Table IV (p. 51) and quoted results obtained by himself and his colleagues, or by Dr. Matthew Young, leading to the conclusion that the fairly steady negative correlation between the death-rate from pneumonia of one week, and the mean air temperature of the previous week (London) was chiefly due to the death-rate in later life and not to that affecting young children. Professor Greenwood also urged the importance of separately considering the rates of mortality in age-groups, and summarized some unpublished results of Miss Hilda Woods, which suggested that the mortality from pneumonia of young children was highly, of adults under 65 significantly, and of persons over 65 hardly at all, correlated with measures of domestic overcrowding, such as the proportion of the population housed more than two to a room. He hoped that Dr. McClure would add to the obligation under which he had placed the Section of Epidemiology and State Medicine by preparing a further, and still more detailed, paper upon the respiratory mortality of Manchester.

Dr. G. CLARK TROTTER (Medical Officer of Health, Islington) referred particularly to the point raised by Dr. McClure in distinguishing between bronchitis, lobar pneumonia and broncho-pneumonia. It was wise to remember that, in a sense, the patient did not die from a particular disease, but died "of the opinion of the doctor last attending," except in the case in which there was a coroner's finding. Thus, as Dr. McClure had indicated, the cases regarded as bronchitis, as broncho-pneumonia, or as lobar pneumonia, were only relatively accurate. This, of course, as far as the paper was concerned, did not matter; some cases went under one classification and some under another, and the error was thus compensated.

It would be interesting to know whether in Manchester there was a scheme for home nursing and, if it could be pointed out, its effect in reducing the mortality. While housing undoubtedly was a potent factor, and the greater mortality could be ascribed to overcrowded conditions, it was necessary to remember that in the better home there was probably better nursing attention given to the case, and that there was a larger percentage of recoveries where the child was nursed and attended to with skill.

The hospital treatment of cases was sometimes unjustly criticized. The hospitals received cases in a fairly advanced stage, and the mortality was necessarily high. On the other hand, when a larger number of cases than was normally accommodated had to be taken into hospital, it was necessary to consider carefully whether the conditions in a crowded hospital were really better than would be obtainable in treatment of a case at home with nursing provided. The removal to hospital of a nervous child might be detrimental, and, in the particular case, such a patient might be better nursed at home. These were exceptional cases, of course, but they were worthy of consideration. On the whole, the majority of cases from the poorer districts were better treated in hospital than at home.

Dr. GEORGE JONES said that the effect of overcrowding depended on the habits of the overcrowded persons; the Jews, for instance, could flourish in conditions under which the English degenerated.

Section of the History of Medicine.

President—Mr. WALTER G. SPENCER, O.B.E., M.S.

The Place of the Tudor Surgeons in English Literature.

By Sir D'ARCY POWER, K.B.E., F.R.C.S.

THE Renaissance in England began with the introduction of printing into this country in the last quarter of the fifteenth century. Before that time manuscripts had been costly and the stationers who copied and sold them were not numerous even in so large a town as London. It was still possible to say whether they had been written by a copyist who came from the north or the south country, and a traveller would have found that the language of the people was a succession of dialects as he passed from one shire to another.

The advent of printing changed everything. The English language was standardized earlier in the written than in the spoken word, standardized, too, in an astonishingly short period of time. The children and the grandchildren of those who had seen the first printed book had developed, as I shall show, a style which in the next generation was polished into the nervous language of Shakespeare, and into the stately cadences of the translators of the Authorized Version of the Bible. Other changes took place in the character of the English nation during this same period. We became a musical race, and for two hundred years nearly every Englishman could take his share in a part song, whilst the Church music and the Graces of the time are still things of great joy when we hear them rendered as they were intended to be given. Cities were small, streets were narrow and houses were stuffy. We had developed the love of fresh air which still makes us offensive to our continental neighbours when they are obliged to travel with us in the same railway carriage, and with the love of fresh air came a love of all that is beautiful in the countryside, the fields, the flowers, the streams and the cattle. All were easy to see and to reach even by the poorest or the most infirm citizen. In London, as soon as he got outside the City walls he was in the country. He could walk on a summer's evening to Clerkenwell, to Bagnigge Wells, to Islington, to Hampstead, or to Paddington, being sure of finding plenty of company in the fields and lanes on the way, and of good refreshment before he turned home again. If he was more pensively inclined and wished for solitude, he could wander along the side of the Tybourne, or along the Westbourne, until he came to Father Thames himself.

My object this afternoon is to show you how faithfully the English surgical writers of the sixteenth century reflect these characters of the English Renaissance. What could be more full of the joy of life than the opening passages of Thomas Gale's *Institution of a Surgeon*, written "at my house in Paddington in the year 1563"? John Yates, a well-known surgeon of the time, is speaking. He begins:—

"Phoebus who chaseth away the dark and uncomfortable night, casting his golden beams in my face, would not suffer me to take any longer sleep but said: Awake for shame and behold the handiwork of our sister Flora, how she hath revested the earth with most beautiful colours, marvellously set in trees, plants, herbs and flowers; insomuch that the old and withered coat of Winter is quite done away and put out of remembrance; at which words of Phoebus my heart quickened in me, and all desire of sleep was eft-soon forgotten. Wherefore I am now come into this beautiful meadow to recreate myself and gather some of those pleasant herbs and flowers which here do grow. But let me see, methinks I see two

men walking together and reasoning also very ernestly. I will approach nearer unto them, perchance they be of my acquaintance; surely I should know them, I am deceived if the one be not my friend Master Gale and the other Master Field. It is so indeed, wherefore I will go and salute them; God that hath brought us together into this place make this day prosperous and fortunate unto you both."

This chance foregathering leads to an interesting and spirited discourse on surgery which, as in the case of Walton's angler, a century later, is thrown into the form of question and answer.

A similar love of Nature is shown by William Bullen, who addresses Thomas Gale as "Amantissimus et Probissimus Vir." He says in his *Dialogue against the Fever Pestilence*, by the voice of one of his characters who is riding to Barnet to escape the plague:—

"How pleasant are these sweet fields garnished with fair plants and flowers: the birds do sing sweetly and pitifully in the bushes; here are pleasant woods. Jesus! man, who would be in the city again? Not I for an hundred pound."

A little later on in the same journey the party is overtaken by a thunderstorm, which is described most graphically. The Citizen first notices its oncoming, saying:—

"Wife, Wife! God send us good luck; do you not see yonder cloud in the West towards the north coming hither. Let us take this house; ride apace: the storm doth begin most fearful. God help us! what shall we do or whither shall we flee? Jesus, Jesus! what a thunder is this. As if heaven and earth should go together. Lord, how the lightning falleth from heaven! All this region is upon a flaming fire; the birds fall from the trees; look how the cattle tremble, and trees are pulled up by the roots, and the houses are burnt with celestial fires."

To which his wife replies:—

"Let us depart from these trees for I have heard say to sit under a white thorn is most safe and surest in a tempest. I have here many goodly jewels against lightning, as the Carbuncle, Emerald, Hyacinthus with Amber and Gold. God and S. Barbara defend us. I have a S. John's Gospel about my neck and a pair of bracelets of Coral about mine arms. Oh God, defend us! I am sorry that we came forth."

William Clowes, writing on the subject of syphilis, gives a good example of contemporary literary style when he says in his preface:—

"I have at this present (loving brethren and friendly reader) to entreat of that disease, and the cure of the same, which in Latin is called Morbus Gallicus or Morbus Neapolitanus; but more properly Lues Venerea, that is the pestilent infection of filthy lust; a sickness very loathsome, odious, troublesome and dangerous. A notable testimony of the just wrath of God against that filthy sin, which at this day, not only infecteth Naples, Spain and France, but increaseth yet daily spreading itself throughout all England and overfloweth (as I think) the whole world and therefore is the cure thereof most expedient, profitable and necessary to be looked unto, And forasmuch as the best avoiding and cure of every disease, consisteth in shunning and removing the cause thereof, I wish all men generally, especially those which be infected, to loathe, detest, hate and abhor that stinking sin, that is the original cause of this infection, and to pray earnestly to God the heavenly Physician and Chirurgeon, for his gracious assistance to the perfect amendment of life; the most safest and surest way to remove it. Secondly, I wish all Magistrates, as the second Chirurgeons appointed of God, even in the love of their country and the zeal and care they have unto their countrymen, to have a watchful eye to find out the offenders in this behalf;—to execute upon them such condign punishment, as may be a terror to the wicked, the rather to abstain and abandon themselves from such abominable wickedness, so filthy in the sight of God and man. Thirdly, the disease being thus ransacked and these causes searched, salved and removed, I doubt not by the help of God, good brethren, I shall here set down a profitable treatise,

with sufficient instructions for the cure of the residue of this sickness so far as my learning and knowledge will extend. In the which I have had no small practice and experience for many years. Let me crave thy friendly acceptation of my hearty good will and faithful zeal to this my country and countrymen whom I see in these days exceedingly afflicted with this noisome and perilous sickness, unto whom notwithstanding I dare promise no help at all, no, not by the best and most sovereign medicines in the world except they be at defiance with this sin and wholly bend themselves to walk in the obedience of God's holy laws. . . . If any error herein have escaped my pen, or if the Printer have made any default; which I suppose some indeed will escape, yea if it were perused and re-perused with Argus eyes; wherefore my good brethren and friendly readers either courteously amend the faults or friendly admonish me of the same, and use so much to thy profit as is found faultless. So shall I find my labours happily bestowed and most willingly accept thy friendly correction."

Every word of this preface resounds with the true spirit of Tudor time. It breathes throughout the newly found spirit of patriotism depending on the fact that England had become a nation. There is a quiet, religious feeling, which was not a pose, and which led simple-minded men to those changes which caused the Reformation. It proves the writer's self-reliance in no spirit of boastfulness, for, as he says, his knowledge is based upon experience. He is clean minded and he clothes his thoughts in excellent English. The personal character of his address shows that London was still a very small place and it is probable that he knew personally the greater number of those who would read his book.

Master Clowes could speak his mind very plainly when the occasion called for it, as may be seen from the following passages, where he is roused by the deplorable state into which English surgery had fallen in his day:—

"Where the learned physician or surgeon cannot be had for council I am herein to admonish the friendly reader in this cause to take heed and not to commit themselves unto the hands of every blind buzzard, that will take upon them to let blood, yea to the utter undoing of a number. For many in these days being no better than runagates or vagabonds do extraordinarily, yea disorderly and unadvisedly intrude themselves into other men's professions, that is to say not only in letting of blood, but also do take upon them farther to intermeddle and practise in this art, wherein they were never trained or had any experience; of the which a great number be shameless in countenance, lewd in disposition, brutish in judgment and understanding, as was their unlearned leader and Maister, Thessalus, a vain practitioner, who when his cunning failed, sent his patients to Libya for change of air. This Thessalus was he which in his own conceit, pleasant fancy or rather foolish imagination said that it was possible to make a perfect physician of a rude man within the space of five months. I will leave him with his fables and riddles, with such other like creaking combatters and slaunderous fellows which persuade themselves to be equal with the best and inferior to none.

This, their grand Captain, was by profession a Teasler of wool and also the fore-runner of this beastly brood following; which do forsake their honest trades, whereunto God hath called them and do daily rush into Physick and Chirurgerie. And some of them be Painters, some Glasiers, some Tailors, some Weavers, some Joiners, some Cutlers, some Cooks, some Bakers, and some Chandlers &c. Yea, nowadays it is apparent to see how Tinkers, Tooth-drawers, Pedlers, Ostlers, Carters, Porters, Horse-gelders & Horse-leeches, Idiots, Apple-squires, Broom men, bawds, witches, conjurers, Sooth-sayers & sow-gelders, Rogues, Ratcatchers, Runagates and Proctors of Spitle houses with such like rotten and stinking weeds, which do in town and country, without order, honesty and skill daily abuse both Physic and Chirurgery, having no more perseverance, reason or knowledge in this art than hath a goose; but a certain blind practice without wisdom or judgement & most commonly useth one remedy for all diseases and one way of curing to all persons, both old and young, men, women and children which is as impossible to be performed or to be true as for a shoemaker with one last to make a shoe fit for every man's foot, and this is one principle cause that so many perish."

This generation of surgeons—Hall, Gale, Clowes and Read—tried an experiment which began and ended with them and, fortunately for us, failed. They essayed to

write in verse. It cannot be called poetry, and very often neither the rhyme nor the rhythm appeals to our ears, although this, of course, may be due to our different method of pronunciation. Hall, of Maidstone, addressed Gale, of London, in this fashion; Gale in turn wrote to Hall; Goodrus and Gerrard produced halting stanzas in praise of Clowes, and Clowes himself was emboldened to epitomize Gui de Chauliac in the same manner. It is not possible to say who began the experiment but it may have been started by Hall, who published in 1550 *Certayne Chapters taken out of the Proverbs of Solomon with other chapters of the Holy Scripture and certain Psalms of David translated into English Metre* with such success that they were attributed to Thomas Sternhold, and in 1565 *The Court of Virtue, containing many Holy or Spiritual Songs, Sonnets, Psalms, Ballets and Short Sentences, as well of Holy Scriptures as others, with Music.* At any rate "the scab soon spread" as one of his contemporaries would have said, and each copy of verses became worse than its predecessor. Here are a few examples in chronological order:—

John Hall writes of the need and conduct of a professional consultation.

When thou art called at any time
a patient to see;
And dost perceive the cure too great
and ponderous for thee;

See that thou lay disdain aside
and pride of thine own skill;
And think no shame counsel to take
but rather with good will

Get one or two of expert men
to help thee in that need:
And make them partakers with thee,
in that work to proceed.

He points out the advantages to be gained both on the part of the surgeon and of the patient and then goes on to say:—

But one thing note, when two or more
together joyned be
About the painful patient
see that ye do agree,

See that no discord do arise
nor be at no debate;
For that shall sore discomfort him
that is in sick estate.

See thou dispraise none other man
his error though thou know;
For sure another for thy plague
shall thee like courtesy show.

Commend the deeds of each good man,
the best, look, that thou may;
So shall good fame redound to thee,
from all men day by day.

And when alone with your foreman,
one of you is present;
Defame nor dispraise in no wise,
the same that is absent.

For nought can more discomfort him
that lies in grief and pain
To hear that one of you doth bear
to other such disdain.

Therefore what so ye have to say
in things about your art;
Let it be done among yourselves,
in secret and apart.

With one consent uniformly
comfort the wounded man;
But unto some good friend of his
express all that ye can

And let them know the danger great
that like is to succeed
Prognosticating wittily
and in convenient speed.

Compare this with Clowes' efforts in the same direction:—

So practice without learning, we ought not to admit,
these two may not be separate that are so duly knit.
There must be a dexterity and a fineness in working,
a quick remembrance eke and a ready understanding.
He must be circumspect and seek to avoid all slander,
not too covetous for money, but a reasonable demander.
Being good unto the poor, let the rich pay therefore,
so God will bless his doings and he shall have the more.
He must also be honest, in living eke upright,
to serve the Lord in truth, he ought to have delight
Avoiding of drunkeness and riot to detest
lest he grow fit for nothing but Bacchus belly feast.

John Gerard, writing in commendation of a book by Clowes, sings:—

Some errors yet if such there be,
Clowes willing mind may quickly mend anew
For wise men wink when often times they see,
Whilst fools are blind when most they seem to view.

Gale is perhaps a little better as a versifier, but not much. Sending out his translation of the *Methodus Medendi* from "my poore house in London the vii of Novemb. 1566," he says:—

Go forth my painful book
Thou art no longer mine;
Each man may on thee look
The shame or praise is thine.

Thou mightst with me remain
And so eschew all blame,
But since thou wouldst so fain
Go forth in God's name.

And seek thou for no praise
Nor thanks, nor yet reward
Nor each man for to please
Have thou no great regard.

Bullein in his *Dialogue bewtween Soareness and Chirurgi* has some very bad lines describing the unqualified surgeons of his day, which begin:—

Some men in misery strange shifts will make
Spending time vainly and labour forsake;

but no attempt seems to have been made to carry on the practice after his time, and he died in 1576.

I think that I have gone far to substantiate the claim I made at the beginning of this short paper, that the Elizabethan surgeons have a very definite place in English literature at a time when our written language was attaining its highest place.

56 Spencer: *Wolveridge's "Speculum Matricis"*

Wolveridge's "Speculum Matricis" (1671), with Notes on Two MS. Copies in the Society's Library.

By HERBERT R. SPENCER, M.D.

WOLVERIDGE'S *Speculum Matricis or The Expert Midwives Handmaid* is one of the rarest books on midwifery; indeed no less an authority than Dr. Aveling feared that what he described as "the earliest original work on midwifery in the English language" has been irretrievably lost.[1] It was *one* of the earliest works on midwifery to appear in English, the first (original) work being the translation (1653) of Harvey's *De Generatione Animalium* (1651). The *Speculum Matricis* has little claim to originality and is not quite so rare as Aveling at first thought, for afterwards[2] he was informed that there was a copy in the Radford Library at Manchester, and another was at Capel, Surrey, in the possession of Mr. John Lee Jardine, who presented it to the Royal College of Surgeons. It is now in the Royal College of Surgeons Library, stamped with the date of its reception, 11 July, 88. It is bound in leather, is smaller than the Radford Library copy, having been somewhat cut down, so that it only measures $5\frac{1}{2}$ in. by $3\frac{11}{16}$ in. It has twenty-eight preliminary leaves, including the two-compartment frontispiece by Cross, and consists of 156 printed pages, pages 144 and 145 and pages 159 to 166 being copied in manuscript from the Radford Library copy.[3] It contains eight plates and twenty-one illustrations in the text. There is no copy of the book in the libraries of the British Museum, the Surgeon-General (Washington) or the Royal College of Physicians, London. There is a perfect printed copy of the *Speculum Matricis*[4] in the library of the Royal College of Physicians, Edinburgh, and so far as I know the only other printed copies (both imperfect) known to exist at the present time are those mentioned, viz., the copy in the library of the Royal College of Surgeons and that in the Radford Library of St. Mary's Hospital, Manchester. Through the kindness of the staff of the latter institution and especially of Dr. Fletcher Shaw, who sent me the book for inspection and has furnished me with a photograph of the title-page, I am able to exhibit it.

The Radford Library copy is a small octavo volume (6 in. by $3\frac{3}{4}$ in.) bound in contemporary leather (somewhat rubbed and worn) and wants the two-compartment frontispiece. It has twenty-seven preliminary leaves, 166 pages, twenty-one engravings in the text and eight separate plates. Although the author in his address to the reader, says that the book was "never intended for the Irish" he published it with the title *Speculum Matricis Hibernicum, or the Irish Midwives Handmaid*, London, 1670. Of this work there is a copy in the Bodleian Library, and I recently saw another copy (with the date torn from the title-page) in a bookseller's catalogue, but was too late to secure it, being forestalled by my friend, Professor Essen-Möller, of Lund, Sweden. Although the *Speculum Matricis Hibernicum* is dated 1670 it seems probable that it was printed after the English edition.

The *Speculum Matricis or The Expert Midwives Handmaid* contains little which is original and I find that for the most part it is a sheer plagiarism[5] from

1 *Trans. Obst. Soc. Lond.*, xxvi, 31.

2 *Brit. Med. Journ.* 1884 (i), 435.

3 J. B. Bailey, *Athenæum*, Nov. 17, 1888, 667.

4 I am indebted to Mr. Graham, the librarian of the Royal College of Physicians, Edinburgh, for particulars of this copy, which is perfect and has the two-compartment frontispiece.

5 Plagiarism appears to have been rife at this period and sometimes led to legal proceedings; as in the case of Rider's Dictionary concerning which Thomas Fuller (*The Worthies of England*, 1662) writes: "Such plagiary-ship ill becometh authors and printers and, the dove being the crest of the stationers' armes, should mind them not, like rooks, to filch copies one from another."

The Expert Midwife, an English translation of Rueff's *De Generatione Hominum* (1580, &c.) which was published in 1637. An appearance of originality is given by casting Rueff's work into the form of a dialogue between Dr. Philadelphos

FIG. 1.—Pen-and-ink copy, by John Lee Jardine, of the Frontispiece of Wolveridge's *Speculum Matricis* (1671). The doctor in the lower compartment is said to be Wolveridge. From the MS. copy in the Society's Library.

and Mrs. Eutrapelia, the midwife. The illustrations also are taken from Rueff, who in turn had copied them from Rösslin (*Rosengarten*, 1513).

The frontispiece of the *Speculum Matricis*, engraved by Cross, appears to be

original. It has two compartments; the upper representing a lying-in chamber with the lying-in patient, the midwife and the infant; the lower representing the doctor (supposed to be the author) wearing wig and hat and addressing the pregnant

SPECULUM
MATRICIS;
OR, THE
EXPERT MIDWIVES
HANDMAID.
Catechiſtically Compoſed,
BY
JAMES WOLVERIDGE,
M.D.
With a Copious Alphabetical Index.

WRITTEN
IVXta MagnaLIa DeI sCrIptor,
Anno Domini, 1669. } *Chronogramma,* · 1669.

Damnoſa quid non imminuit dies?
Ætas parentum, pejor avis, tuli
Nos nequiores, mox daturos
Progeniem vitioſiorem.
Horat. Lib. 3. Carminum, Ode 6.

London, Printed by *E. Okes*; and are to be ſold by *Rowland Reynolds*, at the Kings-Arms in the *Poultrey*, 1671.

FIG. 2.—Pen-and-ink copy, by John Lee Jardine, of the Title-page of Wolveridge's *Speculum Matricis* (1671). The final "t" has been omitted in "tulit" in the *Horatian ode*.

patient and the midwife, who holds a book (no doubt the *Speculum Matricis*) in her hand.

This frontispiece is interesting also because it appears in an anonymous work entitled "*The English midwife, enlarged*, containing Directions for Midwives,

&c., &c., the whole fitted for the meanest capacities. Illustrated with near 40 copper cuts London, Printed for Rowland Reynolds next door to the Golden Bottle in the Strand at the Middle Exchange door, 1682."

The preface to *The English midwife, enlarged*, addressed "to all English midwives," says:—

"You are here presented with an amendment and supplement of what was necessary and yet wanting in this book formerly: so that you will find it to be wholly completed for your purpose, in every respect: it being altogether grounded upon many years' experience and observation in the practice of deliveries: most others being written by those that never practised the art: and some fathered upon persons that were no more concerned in them than the Pope of Rome; such as Sir Theodore de Mayern, Dr. Chamberlen and others, by the Publishers of the *Compleat Midwives Practice*,[1] so that I may justly say of this book, as the learned Sir Richard Baker says of his chronicle, that if all others were not to be found this alone were sufficient, with your diligence &c., &c."

The English Midwife enlarged is copied in great part from Wolveridge's *Speculum Matricis*. The writer even adopts the catechistical method of Dr. Philadelphos and Mrs. Eutrapelia. The order of the chapters is somewhat altered and there are verbal omissions and insertions; but in the main it is a copy of Wolveridge's work, without any acknowledgment of that author. Probably it was in the hands of the publisher Reynolds, who also sold the *Speculum Matricis*, and (? after the death of Wolveridge) thought it was an opportunity for bringing out a new edition. The title suggests that the book had formerly been published as *The English Midwife*, but I cannot find any record of the book bearing that title and the contents of *The English Midwife Enlarged* show that the former book was the *Speculum Matricis*.

The author of the *Speculum Matricis*, James Wolveridge, was an Englishman practising in Cork. Sir Charles Cameron, in his *History of the Royal College of Surgeons of Ireland*, states:—

"His name appears, but with a '?' before it, in Belcher's List of the Fraternity of Physicians, Trinity Hall, Dublin, established in 1660. I find the name 'James Wolveridge, M.D., 1664,' in Dr. Todd's Roll of Graduates of the University of Dublin."

Thus the author was a graduate of but five years' standing at the time of writing the book (1669).

It begins with a chapter by The Author to The Reader, signed James Wolveridge, from my Study in Cork, January 12, $16\frac{69}{70}$.

The book is not intended for the Irish but for the English, "his countrey-folk"; then comes an English ode by Jonathan Ashe e Coll. Oriell, Oxon, "To his deserving friend Dr. James Wolveridge," followed by a Latin ode by the author on his book: to this succeeds an English ode by Aquila Smyth, M.D., "on the Praise, and the happy delivery of James Wolveridge, Dr of Physick, in his labours on the Labour of Women, etc."; then follows an Encomiastichon in Latin addressed to the author by Jonathan Ashe; another, also in Latin, by Daniel Colman; then an ode in English by the author to his book and an Acrostick to Wolveridge by his obliged friend Richard Sampson.

The author reproduces Harvey's account of the unaided delivery of the wife of the soldier on the march, but he does not seem to have read Harvey's work (*De Generatione Animalium*) appreciatively, for he says "from whence it appears the liver to be a congealed and concrete blood"! The chapters on Anatomy and Development of the fœtus, as also the illustrations, are taken from Rueff;

[1] *The Compleat Midwives Practice*, 1656, and *The Compleat Midwives Practice Enlarged*, 1659, by R.C.I.D.M.S.T.B: the second edition corrected: in this edition the names of Sir Theodore Mayern, Dr. Chamberlen and Mr. Nich. Culpepper are mentioned on the title-page.

Section V and onwards contains the Dialogue between Eutrapelia, the Midwife, and Philadelphos, the Doctor.

At p. 78 appears the following:—

Dr. Philadelphos (addressing Mrs. Eutrapelia):

"Having thus run through births, as well natural as præternatural, I shall give you the reason (and that in my own opinion) why these births are of so various and different postures in the womb, observing not alwaies the same posture: and 'tis thus. Because the infant swimming in water and moving itself, sometimes this way, sometimes that way and is bent and tumbled several waies; insomuch, that sometimes it is strangely entangled with its own navil-cord, which I am confident you have seen in your own experience oftentimes."

This is taken almost verbatim, without acknowledgment, from W. Harvey's *Generation of Living Creatures*, 1653, p. 471.

"For he swimmeth in a water, and moveth himself to and fro, he stretcheth himself now this way and anon that, and so is variously inflected and tumbled up and down; in so much that sometimes, being entangled in his own Navel-string, he is strangely insnared."

Wolveridge throws some light on the contemporary management of the puerperium, for he advises:—

"That women be kept in their beds for five daies at least after their delivery. I know 'tis usual for them to rise at three daies; but this to be sure the longer women contein themselves in their bed the more secure they are from danger."

So much for James Wolveridge and his printed book.

The main object of the present communication is to call attention to the fact that in the Obstetrical Society's Library, now incorporated with the Library of the Royal Society of Medicine, we possess two MS. copies of this rare work, one indifferently written and the other, made by one who modestly signs his initials J. L. J., a beautiful specimen of penmanship and draughtsmanship which make it one of the treasures of our Library. I purpose to give some account of the origin and author of this magnificent manuscript copy of a very rare book.

In the year 1884 a copy of the printed work was known to be in the possession of Dr. Fordyce Barker, of New York, and the Obstetrical Society of London had endeavoured to obtain a manuscript copy of the book. But Dr. Aveling reported[1] that:—

"The attempt of the Society to obtain a transcript of James Wolveridge's work on Midwifery, Dublin, 1670,[2] had failed, the man whom Dr. Fordyce Barker had employed to copy the book having absconded[3] with the volume and died in Europe. By this loss of the only known copy of Wolveridge's work it is feared the earliest original work on Midwifery in the English language has been irretrievably lost."

Extracts from Minutes of the Council of the Obstetrical Society of London.

April 2, 1884, *meeting.*

"Two letters were read, one from Dr. Fordyce Barker, thanking the Society for a cheque for £5 sent him to procure the copying of a rare work (*Speculum Matricis*) and stating that the copyist having disappeared with the book and then died (as reported) he had been unable to get it copied; he returned the cheque. A letter from Dr. Wiltshire on the same subject was read."

April 28, 1884, *meeting.*

"Dr. Aveling proposed that a M.S. copy of Wolveridge's *Speculum Matricis* be made and that Dr. Cullingworth be empowered to incur the necessary expense."

1 *Trans. Obst. Soc. Lond.*, 1884, xxvi, 31.

2 It should have been London, 1671.

3 From information furnished by Dr. Clement Godson it appears that the copyist, a Frenchman named Bourgeaud, did not abscond, but died in great poverty in New York before the copying was completed. *Brit. Med. Journ.*, March 1, 1884.

July 2, 1884, *meeting.*

"Dr. Herman read a letter from Mr. J. Lee Jardine, stating his intention of presenting to the Society a MS. Copy of the 'Speculum Matricis.'"

October 8, 1884, *meeting.*

"A letter from Dr. Cullingworth was read accompanying a M.S. copy of the *Speculum Matricis* which he had had made at a cost of £3." (This is evidently the inferior MS. now in the Library.)

June 5, 1888, *meeting.*

"It was proposed by Dr. M. Duncan, seconded by the President and unanimously agreed that the best thanks of the Society be given to Mr. J. Lee Jardine for the beautiful transcript of Wolveridge's *Speculum Matricis* which he had made for the Society, and that a copy of this resolution be sent to Mr. Jardine. It was further agreed that the said transcript be well bound."

Signed: JOHN WILLIAMS.

This manuscript is the volume with which this note is mainly concerned. It is contained in a special case and is of quarto size and bound in whole dark morocco. It is a beautiful example of fine writing and drawing with the pen.

It was evidently a labour of love and seems to have occupied the author's leisure hours for nearly four years.

Extract from the *Medical Directory* for 1884.

JARDINE, JOHN LEE, Capel, Dorking, Surrey—M.R.C.S. Eng. and L.S.A. 1850 (St. Thos's) Fell. Meteor. Soc.: Mem. Path. Soc.: Med. Off. Capel Village Hosp.: late Ass. Surg. Brit. Civ. Hosp. Smyrna, and served with Light Cav. at Eupatoria during Crimean war; formerly House Surg. St. Thos's Hosp., Lond. and Hants County Hosp.

I am able to give some further interesting particulars of the transcriber, John Lee Jardine, owing to the kindness of the Rev. Charles Inge, vicar of St. Giles' Church, Oxford, who was so good as to send me, in reply to my inquiries, the following letter:

"I am very glad to give you information about my dear old friend Dr. Jardine. He was living with some nieces of his wife at Holmwood in Surrey where I was vicar. His nieces married and the home was broken up: so we invited him in 1912 to come and live with us. In the following year he moved with us to Oxford and remained with us till his death in 1921.

"He was one of the best and most charming of men—absolutely unselfish and untiring in his kindness. He was a very faithful and devout churchman and did much for the parish of Capel during his long residence there. Of course I only knew him in his old age: he was 79 when I became vicar of Holmwood; but he retained his vigour of mind and body to the last, in a wonderful degree; taking long walks till he was 90 years of age; and losing neither sight nor hearing. He was no mean artist and I have a delightful album full of his drawings and water-colour sketches made on his many travels and ranging in date from 1846 to 1916. The many sketches made during the Crimean war are especially interesting. He was also musical, with a pleasant light tenor voice. He joined the Holmwood choir when he was 84!"

Mr. Inge adds a chronology of Jardine's life from which I extract the following:—

John Lee Jardine was the youngest child of Alexander Jardine and Sarah (Lee) and was born on Oct. 30, 1827 at Brixton Hill. He had seven brothers and sisters who all died as infants or young children except the first-born, Sarah Lee, who lived to the age of 80. His father Alexander Jardine, born Aug. 20, 1780, died April 30, 1848 aged 68. His mother Sarah Lee, born March 24, 1785, died March 24, 1874, aged 89.

In 1834 J. L. J. was at Eastbourne with Bickerdikes.

1839 went to Warley Court by rail and coach.

1843 went to University College School and was bound pupil to Mr. Ord[1] for five years.

[1] Mr. George Ord, father of Dr. Miller Ord: he practised at Brixton Hill for over fifty years.

62 Spencer: *Wolveridge's "Speculum Matricis"*

1846 entered St. Thomas's Hospital (Oct. 1).
1852 was House Surgeon at Winchester Hospital.
1855 went to Smyrna via Ostend, Cologne, Hanover, Leipzig, Dresden, Prague, Vienna, Laibach, Trieste, Corfu, Cape Matapan, Athens (March 2-19).
In September to Balaclava, Eupatoria till November. 1856-1857 was House Surgeon to St. Thomas's Hospital.
1858 went to Capel, Surrey ("The Cottage").
1863 was appointed Vicar's Churchwarden.
1866 bought Burroughs's practice. On Oct. 2, Capel Church was reopened after restoration and the village Hospital opened by Bishop Sumner of Winchester.
1886 married (Sept. 7) Miss C. E. Morgan.
1900 moved to Dedisham, Rudgwick, Sussex; sold practice to H. Ward Clarke.
1901 went to Braydell Lodge, Wivelsfield.
1903 wife's illness: she died in 1920.
1904 went to Grandon Lodge, Holmwood, the house of his wife's nieces.
1912 to the Vicarage, Holmwood.
1913 to St. Giles' Vicarage, Oxford.
1921 died Nov. 3rd at the beginning of his 95th year and was buried on Nov. 5th in Wolvercote Cemetery, Oxford.

I feel sure the Society will appreciate these biographical details of the author of the Manuscript, kindly given by the Rev. Charles Inge, and will see in them a a reason for the rule for not limiting the Membership of the Section of The History of Medicine to the medical profession.

Section of Laryngology.

President—Dr. ANDREW WYLIE.

Dental Cyst of the Right Upper Canine.

By DAN MCKENZIE, M.D.

THE patient, a woman aged 28, came to the French Hospital with a hard, bony swelling of four months' duration on the ascending process of the right superior maxilla. A small granulation on the labial aspect of the alveolar process in front of the canine socket led by a narrow track into the heart of the tumour. The canine tooth had been removed. Chloroform was administered and the tract was opened up; a cystic distension of the bone was disclosed. The lining was removed, and the cyst cavity opened into the nasal cavity by removal of its inner bony wall. During the operation a free flushing of the right conjunctival sac with tears was observed, but it ceased with the cessation of manipulations.

Specimen Showing Invasion of Endocranium by Carcinoma of Ethmoid.

By DAN MCKENZIE, M.D., and G. SCOTT WILLIAMSON, M.D.

THE patient was a man aged 50 upon whom two operations had been performed for extensive carcinoma of the ethmoid, starting probably near the right orbital wall, as the orbit was invaded by the growth. Death from purulent meningitis followed the second operation. Post-mortem examination showed that the growth had invaded the cranial cavity through the cribriform plate and by way of the perineural lymphatics of the olfactory nerves; that is to say, by the same route as that taken by the bacteria of sepsis in meningitis arising from a nasal focus.

Pathological Report :

The dura was punctured or exposed at operation, the site is in the right fossa, adjacent to the tumour masses on the right olfactory nerve; there, a rounded mass of granulation tissue, overlain by a thin membrane, appears. This mass was not adherent to the pia; similar tissue lines the right frontal sinus, which is opened. There is no malignant growth in this granulation tissue mass and the dura limits the spread of the reaction, but apparently has allowed bacteria to filter through.

Coming to the secondary tumour within the skull, the spread is bilateral. It is progressing directly through the cribriform plates, in the lymphatics of the olfactory pad, travelling thence into the lymphatics on the superior surface of the olfactory nerves, i.e., in the pial extensions on these nerves. Running back in this tissue the growths reach the surface of the brain, just in front of the origin of the nerves, and there the growth presses upon and penetrates (not invades) the brain substance, i.e., the spread is always in the pia—the two pressure necrosis areas in the surface of the olfactory lobes are well shown. Note on the left side the mass is larger—and has become vascularized as the result of the reaction to pressure necrosis. (Note: piece of brain tissue is firmly welded to the mass.)

The instructive features of the preparation are: (1) The lymphatic pial pathway of the olfactory tract. (2) The specific limitation of this track, being clearly marked off from any tracks in the dura mater.

22 Layton: *Tuberculosis of Larynx;* Howarth: *Laryngeal Lesion*

Tuberculosis of Larynx.

By T. B. Layton, D.S.O., M.S.

A. D. B., MALE, aged 48.

November 18, 1925.—Seven months' hoarseness, whole larynx injected. Both V.B.'s swollen and irregular. Interarytænoid space filled up with irregular mass. Vocal cords red, not swollen or ulcerated. Appeared to have pain in swallowing. Advised to go into sanatorium; given advice pending admission. Met casually on December 2, 1925, when he looked so much worse that he was told to go to bed at once and stay there till he got into the sanatorium.

December 18, 1925.—Admitted King Edward VII Sanatorium. Stage II. Right lung + + ; left lung + infiltration.

Larynx (Sir StClair Thomson): Epiglottis congested and infiltrated with ulcer on left border and all laryngeal surface. Ulcerating deposit of left aryepiglottic and of both ventricular bands, largely concealing vocal cords. No dysphagia. Interarytænoid clear. Silence ordered.

February 27, 1926. Clean and subsiding. Posterior two-thirds of vocal cords showed clear.

March 20, 1926.—Vocal cords clear, but anterior quarter concealed by ulcerating deposit on both V.B.'s. Right arytænoid seemed clear, and also left, except that it did not leave and expose the left sinus pyriformis. Interarytænoid clear.

April 15, 1926.—Both vocal cords seen to be intact, though catarrhal and faintly pink. Interarytænoid sound. Both arytænoids enlarged, but mobile and clear. Each sinus pyriformis clear. Much ulceration of epiglottis and both V.B.'s. Galvanocautery (1st) five points epiglottis, two in R.V.B.

May 15, 1926.—Healing, good scar in R.V.B. G.C. (2nd) five points epiglottis, two in L.V.B.

June 19, 1926.—Vocal cords clear. A little thickening over anterior part of L.V.B. Epiglottis very irregular, but healing.

Discharged June 28, 1926. Lung disease improved, quiescent—sputum negative in twenty-four examinations; weight increased from 9 st. 12 lb. to 10 st. 11 lb.

Seen by exhibitor September 17, 1926. Irregularity upper margin epiglottis. General redness including cords, swelling front end R.V.B., which prevented view of front end of vocal cord; no granulations. Advised to continue whispering for a time.

October 5, 1926.—(Sir StClair Thomson): Larynx quite healed, wasted and irregular epiglottis, stump healed, scars on both V.B.'s. Rest normal. Given permission to phonate. The man's courage and implicit obedience were large factors in his recovery.

Laryngeal Lesion associated with Apparent Miliary Tuberculosis of the Lung.

By Walter Howarth, F.R.C.S.

G. B., MALE, aged 21.

Hoarseness for three months. Examination showed a granulomatous patch at the anterior end of the right vocal cord. There was no limitation of movement.

Portion removed for microscopy showed typical tubercle. There were no physical signs of disease in the lung, but the skiagram shows that both lungs are a mass of miliary tubercle. In spite of the absence of symptoms, these cases are invariably and often rapidly fatal.

Discussion.—Mr. F. T. G. HOBDAY said he had seen a cow with a tuberculoma just below the larynx. The animal was killed under the Tuberculosis Order, and was found to have been suffering from the disease in an advanced and generalized state.

Sir STCLAIR THOMSON asked whether Mr. Howarth's case was rightly defined as "miliary" tuberculosis of the larynx. It seemed to be a most chronic and quiescent condition possible, and there was no invasion of the arytenoid region, nor of the epiglottis. Mr. Howarth had informed him (the speaker) that X-ray specialists were now able to tell from the skiagram when a patient had miliary tubercle of the lung. This patient in Mr. Howarth's case had a quiet pulse, he ate well and maintained his weight, and he did not exhibit a raised temperature. It would be a great advantage if the expert radiologists could say definitely that such a man would be dead in a few months. Mr. Layton's case was quite different; it was chronic, indolent and almost lupoid from the beginning. A good prognosis had been given, yet the thorough treatment occupied seven months, and if the patient had not been in a sanatorium, he would probably have gone downhill. It was doubtful whether there had ever been tubercle bacilli in his sputum; they were not found in it while he was in the sanatorium. When he had come under his (the speaker's) observation there was no disease in the interarytænoid or arytænoid regions, but it was extensive in the epiglottis and anterior commissure. When tubercle began in its favourite place, the arytænoid region, and then spread to the epiglottis, the outlook was very serious; but when it began in the epiglottis and the arytænoid was free, the case was, *cæteris paribus*, a favourable one.

Dr. JOBSON HORNE said that when the epiglottis was the only part attacked it was as well to exclude other possible factors from a diagnosis of laryngeal tuberculosis. Miliary tuberculosis—in the commonly accepted pathological meaning of the term—did not occur in the larynx. Diffuse, superficial, minute, discrete, multiple ulcers occurring in the pharynx or larynx in the course of tuberculosis did not constitute miliary tuberculosis.

Tuberculosis of the larynx in animals was very rare, if not unknown; the reason being that the animal either was killed or died before the disease in the lungs had had time to infect the larynx. Now that the specimen of the larynx from a cow had been laid open it was clear that the tuberculoma was below the larynx and in the trachea.

Sir WILLIAM MILLIGAN said it was difficult to explain away the evidence of the microscope, and yet on the other hand it was difficult to decide that this patient had miliary tuberculosis. He (the speaker) had never seen miliary tuberculosis of the larynx, but he had seen more than one case of miliary tuberculosis of the pharynx. Professor Dreschfeld used to teach that the nearer tuberculous disease was to the surface of the body the more rapidly fatal it was. Mr. Howarth's patient, though he presented evidences of tuberculosis in his larynx, was in such relatively good general condition that doubts arose as to the existence of miliary tuberculosis.

Mr. W. J. IBBOTSON said he had had a similar case, one in which the patient had been in a sanatorium and a very good result had been obtained from the intra-laryngeal use of radium combined with ordinary sanatorium treatment. He suggested that radium should be more widely used in the treatment of this disease.

Mr. HOBDAY said he agreed with Dr. Jobson Horne that it was not common to find tuberculosis in the actual tissue of the larynx of animals, but it was often met with in the glands in the region of the larynx of the cow. It was rare in this region in the horse, the dog and the sheep.

Mr. G. W. DAWSON asked how miliary tubercle could be diagnosed by means of the microscope.

Mr. HOWARTH (in reply) said that the case presented some unusual features. The lesion was originally on the anterior end of the right cord. There were no physical signs of disease in the chest, and a piece removed for examination was reported tuberculous. The radiologist said that both lungs were riddled with miliary tubercle and several physicians and radiologists agreed with this diagnosis. This case had followed on two other recent cases, one of which had begun very similarly and had presented almost the same X-ray picture (film exhibited). This other patient had been apparently perfectly well and without abnormal temperature but, later, extensive disease had developed and the patient had died within six months. He (Mr. Howarth) did not profess to be an expert either in pathology or radiology, but many people were apparently certain that this was a miliary condition and that the patient would not live long. Time would show. He would report on the case later.

24 Carruthers: *Swelling on Right Vocal Cord*

Swelling on Right Vocal Cord: Case for Diagnosis.

By N. STUART CARRUTHERS, F.R.C.S.

E. B., FEMALE, aged 25. Works in boot factory.

First seen June, 1925, complaining of hoarseness. Onset had been gradual; patient noticed that it was worse towards the evening. X-ray report: "No evidence of tuberculosis of lungs." Wassermann negative. Physician's report on chest: "No clinical evidence of tuberculosis."

On examination a small isolated nodule was seen in the middle third of the right vocal cord, with minute vessels radiating from it. In spite of rest to the voice the condition has advanced. The greater part of the cord is now involved by an oval swelling, but there is still no clinical or X-ray evidence of pulmonary tuberculosis.

Discussion.—Mr. CYRIL HORSFORD said he regarded this as a common condition frequently seen in singers. The patient said she used to sing, and had noticed that after a vocal effort her voice became hoarse. The condition might be called fibroma; he believed it to be a result of vocal strain; there might have been hæmorrhage in the substance of the cord, and this might have become organized. He (Mr. Horsford) had never tried to treat these conditions, as it was difficult to influence them. The only hope for such patients was a change of vocal method, which would probably relieve the laryngeal irritation.

Sir STCLAIR THOMSON said he thought this was an innocent thickening, fibromatous, fibrocystic, or lymphatic. He would like to hear from those who had tried to treat such conditions surgically whether they had not always failed. At one time the galvano-cautery was used for them. It would be an advantage to have a general agreement as to what was desirable in these cases; whether to leave them alone, dealing only with contributory causes in the mouth, or to treat them by any means other than local surgery.

Dr. JOBSON HORNE considered the condition was vascular in origin and due to strain of voice. He was not in favour of an endoscopic examination, as had been suggested; in the first place, complete rest of voice was indicated.

Sir WILLIAM MILLIGAN said that he disagreed with both the previous speakers, and regarded the swelling as a typical node, with some hyperplasia. It was a great mistake to condemn the use of the cautery in such a case, which, if treated merely by silence, would take ten years to cure. The node could be exterminated by the "direct" cautery, and no risk was attached to that procedure. He (Sir William) had treated many singers, some of them famous, who had suffered from nodes. He had always employed an anæsthetic, and had gently touched the site with the point of the cautery; the results had been excellent.

Dr. ANDREW WYLIE (President) said that at one time he had been enthusiastic about the use of the cautery, but a few years ago two or three very serious complications had occurred and had caused him to change his mind considerably. In one case treated by the cautery, he had been summoned to perform tracheotomy in the middle of the night. A similar event occurred three months later, and since then he had refrained from using the cautery, unless the growth was very small. He thought that vocal rest for fully six months would cure this patient.

Mr. CYRIL HORSFORD said he had seen many cases of this kind, and there was neither harm nor danger associated with them. The only real trouble was in the case of a professional singer, in whom absolute perfection of tone was desired. He did not recommend the use of the galvano-cautery in that case. He had seen this condition in professional singers, and it was most difficult to cure. The nodule was not on the edge but in the substance of the cord, therefore one could not interfere with it surgically by any cutting process, and it was not removable by cautery. A nodule on the edge of a cord might be touched lightly by the cautery, but a nodule in the centre so treated would be replaced by a scar, which would distort the cord and spoil its value for singing.

Sir JAMES DUNDAS-GRANT said that the cautery was useful in the case of nodules found at the junction of the anterior and middle thirds of the cord. He had a case supporting this view, that of a woman with a large family who had to use her voice a good deal. In the present case there was paresis of internal tensors, and vocal treatment should be given.

Mr. LAWSON WHALE said he agreed that there was paresis of the internal tensors in this case.

Mr. CARRUTHERS (in reply) said that this patient had not been kept absolutely silent, but for seven months she had not spoken above a whisper. In spite of this treatment the local condition had spread and she was very hoarse. Perhaps absolute silence would produce improvement. The patient worked in a dusty atmosphere in a factory where there was machinery and it was necessary for her to strain her voice in order to make herself heard.

Basal-celled Carcinoma of Larynx: Recurrence after Five and a Half Years.

By WALTER HOWARTH, F.R.C.S.

MRS. D., aged 58.

Operation: On June, 1921, the whole of the left ventricular band and the left vocal cord along with the anterior third of the right cord were removed. The case was fully reported in the *Journal of Laryngology*, June, 1922.

At the present time there is a small recurrence of the growth on the left side anteriorly.

Discussion.—Mr. LIONEL COLLEDGE said that before the operation the growth had extended to the opposite side of the larynx: therefore, if this was a genuine recurrence, he thought the whole front of the larynx on both sides—possibly even the entire larynx—would have to be removed. If the patient opposed an extensive operation, radium would be worth a trial, as this form of growth genuinely responded to its use.

Sir JAMES DUNDAS-GRANT said that if the patient were not opposed to operation, a portion of the growth might be removed for microscopical examination. But in view of opposition, it might be better not to take a piece away, as the procedure might conceivably stir up the trouble. Basal-celled carcinoma responded favourably to radium treatment.

Mr. HOWARTH (in reply) said that the patient was only opposed to an extensive operation which might involve loss of voice. He proposed to examine her in hospital by the direct method, and to remove a portion of the growth so as to ascertain whether it was a true recurrence or not. He thought more than laryngo-fissure would be necessary, because of the extent of the primary growth, even though it was a very slow-growing type of cancer, practically a rodent ulcer. If the patient would consent to operation he would expose the tumour and imbed radium in it, or carry out a limited excision.

Two Cases of Chronic Hyperplasia of Superior Maxilla.

By WALTER HOWARTH, F.R.C.S.

Case I.—PATIENT, female, married, has noticed for the past four or five years a gradual swelling of the right upper jaw, quite painless in character.

The diagnosis rests on the long history, the minor character of the pain and the resultant deformity, together with the normal condition of the mucous membrane and absence of inflammatory phenomena, especially tenderness on pressure.

Macroscopic and microscopic appearances of a piece of bone removed show the typical structure associated with this condition, which was first described by Mr. Westmacott. X-ray examination shows areas of hyperplasia in the basisphenoid and also in the vault of the skull.

26 Howarth: *Endothelioma of Antrum apparently Cured by Radium*

Case II.—Patient, male, aged 19. Operated on three years ago. The macroscopic appearance of the bone was similar to that in the preceding case, but the microscopic slide shows alternate layers of bone and cartilage, without any fibrous tissue. This appearance is quite different from the usual picture of spongy bone formation with proliferating osteoblasts, together with the destruction of the spongy bone by masses of osteoclasts, a vascular interstitial tissue and the laying down of much fibrous tissue.

The previous operation was not by any means complete, but the clinical appearance has not progressed except in the alveolar border.

Specimen of Hyperplastic Bone from the Frontal Sinus.

By WALTER HOWARTH, F.R.C.S.

THE macroscopic and microscopic appearances present many features similar to those associated with chronic hyperplasia of the maxilla.

Endothelioma of Antrum apparently Cured by Radium. Recent Local Recurrence after Three Years.

By WALTER HOWARTH, F.R.C.S.

MR. C. D. had an enormous tumour of the right upper jaw and was first seen in a moribund condition. Radium produced an almost magical disappearance of the growth. Recently two small nodules have appeared.

Discussion.—Dr. W. H. KELSON, referring to the case of the boy with a swelling of the upper jaw, said that he had seen a number of such cases, and asked whether the trouble might not have started in the nasal cavity. This might help to solve the ætiology, which at present was very obscure. Did Mr. Howarth think that the conditions as shown by the microscopical slides were different stages of the same thing? Professor Knaggs considered them to be probably different varieties of the same disease. This boy appeared to have good teeth, but there was dental trouble in most of Mr. Westmacott's cases.

Mr. W. S. SYME said he had seen numbers of these cases, and did not think that X-ray examination of the sinuses was sufficient to exclude the possibility of sepsis, which was almost always present. These cases ought always to be thoroughly investigated from this point of view.

Mr. A. J. M. WRIGHT said that in one such case he had removed a considerable portion of bone and opened the antrum. In that case the lining of the antrum had been healthy.

Mr. HOWARTH (in reply) said that he had hoped to obtain suggestions as to treatment. Mr. Westmacott, in 1913, had suggested chiselling away all the redundant bone until healthy bone was arrived at. That had been done in the case of the boy (Rouge's operation); the woman had been treated with radium. He thought this might produce a good result, because it was difficult, when doing a Rouge's operation, to be certain where pathological bone ended and normal bone began, as one shaded off into the other. The deformity in these cases was sometimes very distressing. The boy was not cured, but his condition was much improved. He (Mr. Howarth) understood that these conditions did not originate in or invade the nasal cavity, and the remaining portion of the antrum was found to be healthy. The origin of the condition was, he believed, in the teeth, the sepsis there causing a chronic osteomyelitis. Pathologists were not agreed as to the exact nature of the condition.

The specimen of hyperplastic bone from the frontal sinus looked like the same under the microscope. He had shown the case of endothelioma of the antrum in order to demonstrate what could be done with radium in an inoperable growth. It dropped out after radium treatment. There were two small recurrences, which he intended to deal with in two days' time by excision, as radium used for recurrences was in his opinion often unsatisfactory in result.

Section of Laryngology 27

Osteoma of Frontal Sinus.

By D. F. A. NEILSON, F.R.C.S.

MALE, aged 43. First seen 1925. He then consulted his doctor on account of pain and inflammatory swelling over the left eye. This lasted for two weeks, subsided, and has not recurred. For some years previously he had noticed a swelling in the middle of his forehead.

No history of headaches or of frontal pain except during the attack mentioned Nasal fossæ appear normal.

Since 1925 his sight, which, before that date, was perfect, has deteriorated rapidly. The fundi have been examined several times and reported as normal, the defective vision being due to marked astigmatism and myopia. Stereoscopic X-ray films [shown] demonstrate an osteoma with well-defined margin and surrounded in places by the expanded walls of the frontal sinus. The cranial fossa is invaded to quite an appreciable extent, and in this locality the roof of the sinus is well away from the margin of the osteoma. Presumably a mucocele is developing.

The patient has refused treatment, but the rate of increase in size of the growth is being observed.

During the first year no increase was detected.

Sir JAMES DUNDAS-GRANT asked how the defect of vision and the development of polypi in the right nasal cavity could be explained in this case. It seemed as if there had been some invasion of the posterior ethmoidal cells, involving the oculo-motor nerves.

Two Specimens of Exostosis arising in the Floor of the Frontal Sinus.

Shown by WALTER HOWARTH, F.R.C.S.

BOTH these tumours produced a mucocele of the frontal sinus. They are shown as a corollary to the foregoing case.

Tumour of the Nose.

By G. W. DAWSON, F.R.C.S.I.

MALE, aged 24.

There is a tumour on the right side of the nose, extending from the eyebrow to the ala of nose. It is soft and ill-defined and has been present for sixteen years.

Discussion.—Sir JAMES DUNDAS-GRANT said he thought the growth was a lymphatic nævus.

Dr. W. HILL said he would treat it with the ionization needle.

Mr. A. J. M. WRIGHT said electrolysis might be worth trying here.

Mr. M. VLASTO said that as the condition was obviously benign and as, apart from the disfigurement, the swelling did not incommode the patient, he would not advise operation. As an alternative diagnosis, he suggested that of a soft lipoma.

Miss ELEANOR LOWRY said that a few weeks ago she had had a patient with a soft swelling over the mastoid, which, it was said, varied in size. There was a chronic discharge from the ear, and, on operation, the swelling was found to be a lipoma. Possibly the present case might be of the same nature.

Dr. IRWIN MOORE said he, too, thought the swelling was a lipoma, and would advise leaving it alone.

Two Cases of Laryngectomy: Specimens Shown.

By W. J. HARRISON, M.B.

Case I.—MALE, age 54. Complained of weakness of the voice and huskiness of about three months' duration. On examination the right cord was found to be motionless and the posterior third had a "mouse-nibbled" appearance. The right arytænoid was

28 Harrison: *Two Cases of Laryngectomy: Specimens Shown*

immobile and there was a good deal of swelling in this region, extending into the interarytænoid space. Wassermann reaction weakly positive. Pathologist's report on specimen: "A typical squamous-celled carcinoma." The patient is edentulous. Operation, July 20, 1926: Laryngectomy without a preliminary tracheotomy. Larynx removed from below upwards. Uneventful recovery. Primary union of the wound. Highest temperature 99·6. Fastest pulse-rate 112. Greatest number of respirations per minute 24. Now speaks in a whisper, which is becoming louder and more distinct. Has put on weight since the operation (he says 16 to 17 lb.). Specimen opened from the front.

Case II.—Female, aged 37. Had noticed an alteration in her voice for six months, she had taken little notice of it. Had had attacks of weakness of her voice for the past two years.

On examination a fungating growth was found in the upper part of the larynx, on the right side, and a gland on that side of the neck was enlarged. Wassermann negative. Pathologist's report on specimen: "A squamous-celled carcinoma." Patient wears complete dentures. Operation January 4, 1927. Laryngectomy without preliminary tracheotomy. Epiglottis removed owing to situation of growth. The division of the muscles was made some distance from their insertion, as the gland over the cricothyroid membrane was enlarged. The gland in the neck and the gland-bearing tissue in this region were removed.

Considerable difficulty was experienced in closing the pharyngeal wound owing to the free removal of the larynx.

Some sutures in the skin have given way, but healthy granulations are springing up and the wound is closing rapidly (January 20, 1927).

General condition very satisfactory. Temperature once reached 100° F. (on the sixth day after operation). Highest pulse-rate 114, for a short time. Respirations 24 per minute for the first twenty-four hours, since then 22 per minute. Specimen opened from behind.

Discussion.—Mr. HARRISON said that the method he had adopted in dressing these cases was to wrap the tracheotomy tube with gauze so that it completely filled the lumen of the trachea. Ribbon gauze was packed from below upwards into the wound and a piece of gauze was packed in below this immediately above the trachea. These packs had to be changed frequently, at first at about twenty-minute intervals. He wished to learn what other methods Members used. He got such patients up in three days, sitting in an armchair. He would welcome suggestions as to how long the nasal feeding tube should be retained in use in cases which were doing well. He had seen it stated that these patients generally died within three years of the operation, from lung complications. What was the experience of others in regard to lung complications?

Sir STCLAIR THOMSON said that these interesting specimens were very instructive. In similar cases, in early days, he (the speaker) had attempted laryngo-fissure, and usually there had been a recurrence. The kind of case from which these specimens were shown was unsuitable for laryngo-fissure, but was usually admirably suitable for laryngectomy. In the case of the male, it was remarked that the cord was motionless, and his (the speaker's) statistics showed that recurrence, after laryngo-fissure, was much more frequent in cases in which the cord was fixed, than in those in which it was mobile. Mr. Harrison had told him that what was remaining of the cord in the second case had been quite concealed. Mr. Harrison had acted rightly in performing laryngectomy, instead of splitting the larynx or doing laryngo-fissure, but if Mr. Howarth had to operate further on his patient, he (Sir StClair) suggested that it would be worth trying to save part of the larynx, however small, even if a tracheotomy tube had to be worn for the remainder of life. He (himself) had had one case in which he had performed secondary laryngo-fissure. That was three years ago, and the patient was going about in society; the trachetomy tube in her neck was concealed, and her voice was certainly better than none at all.

With regard to these people continuing to breathe through the neck, there was no particular danger in that. In his (the speaker's) book there were references to people who, having

undergone tracheotomy at the age of 30, had died aged 80, and had worn the tube during the intervening time. Despite the *a priori* physiological arguments as to the importance of warming inspired air in the nasal passages, he knew of people who, while wearing their tracheotomy tube, played tennis, begat children, and did all the other things the ordinary citizen accomplished, except swim! Moreover, they did not seem more subject to bronchitis than were other people. The malignancy in Mr. Howarth's case was of a low form, and he (the speaker) thought that an operation might be performed, which, as regarded severity, would lie somewhere between laryngo-fissure and complete laryngectomy.

Mr. C. A. SCOTT RIDOUT said that he had shown at a previous meeting a patient with a similar condition. This patient had progressed well and was quite cheerful, having had no trouble at all, though since the operation he had suffered from an acute attack of appendicitis. He had never had a cold in the head since the throat operation.

Mr. LIONEL COLLEDGE said that in the first of these specimens the epiglottis had been left; in the second it had been removed with the larynx. On close examination the growth was seen to approach closely the cut edge of the epiglottis. It was better, when removing the whole larynx, to take away the epiglottis at the same time. In some of his own cases, on examining the patient with the mirror, the epiglottis had appeared to be free, but afterwards, in the specimen, it was seen to be deeply infiltrated. With regard to drainage, he had not found gauze drains efficient. He used rubber tubes instead, and as soon as there was much discharge he syringed the tubes through with eusol; a simple rubber tube having lateral holes was the best form of drain.

Sir JAMES DUNDAS-GRANT said that in a case in which he had seen Mr. Lionel Colledge operate, the existing tracheotomy wound was adherent to the surface of the neck, and Mr. Colledge had cut through the trachea above it, bringing the skin together behind the cut surface of the trachea, so that he was then able to plug it until union was established and so to protect the lungs from infection—the patient breathing through the tracheotomy tube. Might not this safeguard be more frequently adopted?

Mr. LIONEL COLLEDGE (in reply to Sir James Dundas-Grant) said that a preliminary tracheotomy allowed the cut end of the trachea to be packed so that the trachea was completely shut off. But in three cases in which there had been preliminary tracheotomies there had been recurrence in the tracheotomy wound, and he wondered whether making an additional wound there before doing the main operation did not expose the patient to an additional risk of recurrence.

Dysphagia for Three Months due to Foreign Body in the Œsophagus.

By C. GILL-CAREY, F.R.C.S.Ed.

History.—Patient, a woman of 32, complained of inability to swallow solid food for three months. Could not remember having swallowed a foreign body. Had lost 2 stone in weight.

Examination.—No abnormality in larynx; no excess mucus in the pyriform fossa.

X-ray examination by Dr. Graham Hodgson, who reported an abnormal opacity in the œsophagus between the sixth and seventh cervical vertebræ.

Œsophagoscopy.—Piece of glass, a portion of which is shown, removed from the œsophagus 25 cm. from incisor teeth. No ulceration of the œsophagus was present.

Complete recovery after removal of the foreign body.

Dr. ANDREW WYLIE (President) said that he had seen the patient in this case, and she had complained very little. Mr. Gill-Carey had removed the foreign body in a very skilful manner. It was uncertain at the time of the operation whether a foreign body was present.

Lymphosarcoma of the Tonsil and Tongue with Glandular Involvement.

By NORMAN PATTERSON, F.R.C.S.

PATIENT, aged 64, female.

History.—Eight years ago the patient noticed swellings on the left side of the neck. Later on she was treated at the London Hospital by X-rays, and the enlargement of

30 Patterson—Kelson—Dawson—Macleod

glands disappeared after six months. About two years ago the swelling re-appeared, but the condition improved under treatment. The glands began to enlarge again last summer. The patient has lately been under medical treatment.

On examination there is found to be a large, smooth, firm mass in the region of the left tonsil and there is a similar swelling involving the lingual tonsil. There are enlarged glands on both sides of the neck, and the pre-auricular gland on the left side is distinctly swollen. There is also enlargement of a small gland in the right axilla.

Microscopic Diagnosis.—Lymphogranuloma (so-called large-celled lymphosarcoma). Slide shown.

Discussion.—Sir JAMES DUNDAS-GRANT asked whether radium might be used in this case.

Mr. PATTERSON (in reply) said that the growth had become somewhat generalized.

Growth in Larynx.

By W. H. KELSON, M.D.

H. J., AGED 32, male. Noticed that he became hoarse intermittently soon after demobilization in 1920. Has never had to strain his voice.

Larynx shows general congestion. A roundish, pinkish, mobile swelling, about ¼ of an inch in diameter, can be seen in the region of the anterior commissure attached to the right vocal cord. Tonsils enlarged, but no enlarged glands noticed. No history of syphilis. No tubercle bacilli found in sputa.

Mr. E. WATSON-WILLIAMS thought this might be a granuloma. He had seen five such cases, all in pensioners. When examined after removal, there was no fibrous structure, it was granulation tissue. This growth should be easily removable with the forceps.

FURTHER REPORT OF CASES PREVIOUSLY SHOWN.

Sarcoma in Cricoid Region.

By G. W. DAWSON, F.R.C.S.I.

PATIENT, aged 58, female, married. (Shown at meeting held December, 1926.)

History.—Tightness of throat began last Christmas; later difficulty in breathing and laryngeal stridor. Has been treated for asthma. Wassermann negative. There is a smooth, round tumour covered by normal coloured mucous membrane, situated below the right vocal cord. Both vocal cords and arytænoids move well. Voice normal.

December, 1926: Urgent tracheotomy. Owing to slight bronchial trouble operation postponed to January 4, 1927. Laryngo-fissure. Round-celled sarcoma in cricoid region. Secondary hæmorrhage eight days later. Patient died January 15, 1927.

Mr. DAWSON said the general view, when this case was shown, had been that the growth was an enchondroma. It was a soft growth in the region of the cricoid. He had split the larynx, and it did not appear white, as it had done on laryngoscopic examination. He excised a piece, and the report was that the growth was round-celled sarcoma. The patient went on very well for eight days, then secondary hæmorrhage occurred, and was repeated, and on the following day he died.

Perforation of Hard Palate.

By A. L. MACLEOD, M.B.

MALE, aged 69. The patient has worn a tooth-plate for years. For five years liquids and soft foods get into the right nostril. There is a perforation of the hard palate which the exhibitor suggests is due to pressure atrophy. There is no sign of a neoplasm, but in view of the possibility of malignant trouble, he would be reporting himself every month.

Section of Medicine.

President—Dr. HUGH THURSFIELD.

DISCUSSION ON MIGRAINE.

Dr. C. P. SYMONDS.

THE subject we are to discuss is one about which much has been written and said in recent years. It held, for instance, the place of honour at the Annual Meeting of the Neurological Society of France, in 1925 [18] and was debated at the last Annual Meeting of the British Medical Association [2]. Such popularity should mean that there have been recent important additions to our knowledge. But from a perusal of the literature I cannot find that this is the case. There has been a good deal of debate and speculation but little methodical investigation.

I have no original observations to put before you this evening. The clinical features of migraine are so well described by Liveing [10] in his book published in 1873 that it is difficult to find any aspect which has not been accurately recorded in his pages.

I propose first to plead for a clearer definition of the subject, then to hold up to critical review the main facts and theories at our disposal, and finally to suggest certain lines along which further research should proceed.

The lack of progress in our knowledge of migraine is partly due to faults in our technique of observation. We have been using too low an objective and trying to take too much into the field. There is a tendency to include under the term "migraine" any obscure form of recurrent headache. This, I think, is a mistake. We know very little yet about the mechanism and ætiology of headaches in general. It is true that in recent years there have been notable advances in our knowledge of intracranial physiology, particularly that of the sources and flow of the cerebro-spinal fluid, and the application of this knowledge has provided a basis for clearer thought and fresh speculation. Through it we have been able to remove from their former obscurity the headaches which, without any objective sign of organic disease, may follow even minor injuries of the head. In another quarter the rhinologists, particularly Sluder, have defined a group of headaches which they believe to be due to disordered function of the nasal sinuses. Both these forms of headache are paroxysmal and recurrent, and may be associated with nausea, and might be—probably have been—at times regarded as migraine. I believe that in this direction there is a large territory still unexplored and that many kinds of headache still await proper classification. Their common feature is the recurrence of attacks over a long period without any evidence of organic disease. To include all these under the head of migraine may be convenient at times. It gives the physician a convenient "label" for his patient. But for the purposes of scientific investigation we should begin by reserving the term "migraine" for a group of cases which in

their clinical characteristics are so well defined that we should have no difficulty in separating them from the main body of obscure recurrent headaches.

I propose, therefore, to define migraine as a liability dating from early life to recurrent attacks of headache, which are at one time or another associated with a characteristic visual or sensory disturbance and are frequently accompanied by nausea or vomiting. This definition excludes a number of cases of recurrent sick headache, which we should all probably regard as incomplete migraine, but if we adhere to it we shall be certain that we are all talking about the same thing. It will be one thing to say that eye-strain or the ingestion of hot buttered toast may cause sick headaches, it will be quite a different thing to say that they cause migraine. The narrower definition will also help us to focus our attention upon a few outstanding clinical facts instead of diffusing it over a large field whose details are obscure and whose boundaries are uncertain.

ÆTIOLOGY.

In the ætiology of migraine, as thus defined, there is one outstanding factor generally recognized as of first importance—heredity. It is the rule rather than the exception to obtain a history of direct inheritance, and a Danish writer quoted by Christiansen [18] has recently offered proof that migraine is a dominant hereditary factor in the Mendelian sense. But the attacks are occasional. Given the inherited liability, what are the precipitating causes? Most writers from the time of Liveing onwards have put first in this category mental stress and fatigue. With this I emphatically agree. To the question, "Have you found any particular circumstances likely to bring on an attack?" the commonest answer is "worry" or "excitement." As to the other occasional factors, there is much diversity of opinion. Much has been written and said about the importance of diet. Fothergill [10], one of the earliest writers upon the subject and a sufferer himself, was convinced from personal experience that fats were responsible, and of butter he says, "nothing more speedily and effectually gives the sick headache." This observation has been often repeated and has been brought again to notice in some recent correspondence in the *British Medical Journal*. Marmaduke Fawkes [6], in particular, has drawn attention to the appearance of acetone in the breath and in the urine when an attack is impending, suggesting that there is some defect in fat metabolism as the cause. The withdrawal of fats from the diet and their replacement with glucose together with large doses of alkalis, he states, is often successful in averting the crisis. Allison [1] on the other hand, claims that an excessive sugar intake is the important cause and that elimination of sugar from the diet is frequently successful in prevention. Curtis-Brown [4] considers that the sole cause of migraine is an inherited or acquired defect in protein metabolism. This defect is, in a sense, selective. That is, in one patient it may be beef protein and in another mutton protein, which is imperfectly metabolized and leads to the formation of toxic by-products. The attacks can be prevented by putting the patient first on a protein-free diet and then building it up gradually, eliminating any particular food substance which causes an attack. In criticism of these observations it must be remarked that none of these writers presents any detailed instance of an attack of migraine, coming within our clinical definition, which has been investigated and treated on the lines suggested by them. A few biochemical investigations have been published upon these lines, but with no very conclusive results. Pagniez and Nast [13], however, have recorded a case described as a typical migraine with visual disturbance, in which the attacks were provoked, as a rule, by the ingestion of chocolate. This was followed by a hæmoclasic crisis and after a delay of some hours the attack developed. The liability to attacks could be notably diminished by giving the patient

a dose of peptone by the mouth an hour before meals. This, as a single observation, is suggestive. It has also been held that the attacks are due to acidosis, and may be averted by giving large doses of alkali. On the other hand, Weismann-Netter [16] has arrived, by means of biochemical investigations, at an opposite conclusion. Making serial observations upon the pH and alkaline reserve in two cases, he found that during the free interval the figures for these were normal, but that forty-eight hours before the attack in each case a marked alkalosis developed, the pH, for instance, rising from 7·36 to 7·5, and the alkaline reserve from 59·1 to 73. Paullian [14] has recorded an excess of cholesterin in the blood preceding the attacks. In criticism of these and other such observations which I have been able to discover, it must be said, in the first place, that the clinical data are meagre or unsatisfactory, and secondly, that the biochemical observations have been few in number. The mere occurrence of gastro-intestinal disturbance during the early stages of the attack cannot be taken as evidence of a gastro-intestinal origin.

Of eye-strain I would say that in the particular group of cases we are discussing it is rarely an important cause of attacks. I believe there are recurrent headaches due to errors of refraction and muscle balance, which in many cases can be cured by proper correction, but these should not be included under the head of migraine.

Symptoms.

I have made it a part of my definition that at one time or another the attacks shall have been associated with certain characteristic visual or sensory disturbances. These phenomena are worth studying carefully not only for their diagnostic value but for the light or darkness they may throw upon the vexed question of pathology.

The true fortification spectrum leading on to a homonymous defect in the visual fields is comparatively rare. Much more commonly we meet with a simple defect which may or may not be preceded by vague flashings. This defect may vary from a mere dimness or mistiness of vision to a complete loss. It is usually restricted to a part of the visual field which can be accurately described by the sufferer in answer to leading questions. Usually the defect is a vertical homonymous hemianopia. In the same patient it may be right- or left-sided in different attacks. Sometimes it is definitely quadrantic. It may in some attacks be bilateral, resulting in complete temporary blindness.

There is, however, another less common variety in which the visual fields are divided transversely. Such transverse hemianopia may occur on some occasions in a case in which the more common vertical defect is the rule.

As yet another variant we meet with transient central scotoma, usually bilateral, sometimes affecting one eye only. Finally we may encounter monocular blindness. It is characteristic of all these disturbances of vision that both onset and recovery are rapid and that recovery is complete.

The sensory phenomena are equally distinctive. A sensation of numbness or tingling, beginning in one or sometimes both hands, or in the tongue or lips, spreads slowly from hand to mouth or vice versa. The sensation is similar to that observed in the sensory form of Jacksonian epilepsy, but there are several distinguishing features. The spread in epilepsy takes a matter of seconds or, at most, minutes. In migraine it continues from a time varying from ten minutes to half an hour. The tingling of tongue and lips in migraine is always bilateral, in epilepsy never. Nor are both hands ever affected in an epileptic attack. If the right hand is affected in one of these attacks of migraine there is frequently some disturbance of speech of an aphasic nature. The lower limb is rarely involved.

The sensory and visual disturbances may appear in combination, the one

preceding the other: more usually they occur singly. As a rule the visual or sensory disturbance precedes the headache but may accompany it. It is often associated with only a small proportion of the headaches in an affected person. It may occur without the headaches as an isolated phenomenon.

The headache is of a throbbing, bursting or splitting character. It is commonly unilateral and frequently on the side opposite to a homonymous hemianopia. It is almost as frequently bilateral. If severe it is commonly associated with nausea and vomiting. Both nausea and vomiting, however, may occur in the presence of slight headache, and, in children especially, vomiting may be the predominant feature of the attack. Many of the attacks known as cyclical vomiting in children appear to be predecessors of true migraine. It is worthy of remark that the attacks usually commence in the early morning and the patient often wakes with the knowledge that his attack has begun.

Some persons have a preliminary warning in the form of a peculiar sense of lassitude, or, less often, of extreme well-being the day before an attack is due. Such are the main facts familiar to us all which I have thought it well to recapitulate before embarking upon the difficult problem of

Pathology.

The headache is of a kind frequently met with in conditions of increased intracranial pressure, but I would submit that our knowledge of the mechanism of headaches in general is not yet so exact that we may argue from this that increased intracranial pressure is the cause of the headache in migraine. We have therefore to study the other phenomena and it will be profitable first to inquire whether from the data at our disposal we can localize the seat of the disturbance to any part or any level of the brain or brain-stem.

We may take as our starting point the common vertical homonymous hemianopia. This might conceivably be due to a disturbance of function anywhere between the optic chiasma and the occipital cortex. There is one point, however, in favour of a cortical situation—the hemianopia of migraine is frequently preceded by flashes of coloured light. This symptom is not met with in organic lesions of the optic chiasma, tract or radiation, but does sometimes occur with lesions of the occipital cortex. From this point we turn naturally to the sensory phenomena. The right-sided attacks with aphasia are again suggestive of a postcentral cortical situation. There are, however, several difficulties to be met. If the disturbance is cortical, how is it that the tingling in lips and tongue is always bilateral, and that in the hand sometimes so? If we are going to adhere to our cortical localization we must assume that there are two separate areas of disturbance on the two sides of the brain which are, in the instance of tongue and lips, remarkably symmetrical and simultaneous.

Turning now to the other visual phenomena it is conceivable that the transverse hemianopia might be due to symmetrical bilateral disturbance situated above or below the calcarine fissures. Since the macula has cortical representation it is also conceivable, though less likely, that the bilateral central scotomata might be due to symmetrical disturbance at the occipital poles. But what are we to make of the unilateral scotoma or monocular blindness which is said to occur? The imagination cannot be stretched to include this phenomenon within the hypothesis of cortical localization. We should, therefore, be critical of such instances and inquire whether they are indeed within the limits of our definition of true cases of migraine.

I have notes of one such case only, which, though I made the clinical diagnosis of

migraine, ought not strictly to be included, as up to date the visual phenomena have not been accompanied by headache.

The patient, aged 22, is a sister of one medical man and the secretary of another. About three years ago she first noticed occasional attacks in which a quivering luminosity appeared on one side of objects looked at; sometimes to the right, sometimes to the left. The attacks would last about half an hour, usually occurred in the early morning, and were not attended by any other disagreeable sensation.

As separate phenomena she has had two other kinds of attack. In one variety everything she looked at had a black spot in it, though she could see everything all the way round; in the other kind little lighted matches would float down in front of her eyes. Both forms of attack lasted usually half an hour and were apt to occur in the early morning.

These attacks had appeared to her of so trivial a nature that she did not think of seeking medical advice until she had the following experience: About 10 a.m. one morning at her work she noticed that, as it were, something had been clamped over the left eye. She shut the right eye and found she could not distinguish between light and darkness. Then after ten minutes the blindness went half way up like a blind being drawn up. For a minute it stuck at the horizontal level, then rolled right up. The whole attack lasted twenty minutes.

She was examined by an oculist a day or two later, who found nothing amiss apart from a slight error of refraction. There were no signs of any organic nervous disease. None of her attacks were associated with headache or nausea.

As I have said, the absence of headache in this case precludes its acceptance as a definite instance of migraine, although the story of the visual phenomena is suggestive. Writers on the subject of migraine, however, commonly refer to the recurrence of monocular blindness in true migraine. I have not met with a well documented instance in the literature. Perhaps some of you can make good this defect from reading or personal experience. The point seems to be one of importance, for the occurrence of monocular blindness would exclude any conception of migraine as a purely cortical disturbance.

We may conclude that with this possible exception the *situation* of the disturbance in migraine lies in the cerebral cortex, particularly in those parts in which vision and sensation are represented, and that this disturbance is often bilaterally symmetrical.

Having arrived so far we have to inquire what is the *nature* of the disturbance. Here we are on much more difficult ground, for the cortex is beyond our ken. We have, therefore, to depend largely upon analogies from similar disturbances in other parts. The theory of vascular spasm is an old one and, I would submit, the most probable. In the first place it offers an adequate explanation of the order in which the visual phenomena appear. We are familiar with the principle that minor degrees of anoxæmia lead first to exaltation and subsequently to depression of nervous function (e.g., cerebral anoxæmia of aviators); in the second place it offers the most intelligible explanation of the frequent symmetry of the disturbance on the two sides, for which we have a striking analogy in Raynaud's disease. In this latter affection, which we know from observation to be due to transient vascular spasm, it is common to find simultaneous involvement of corresponding digits in the limbs of the two sides. In this connexion it is worth dwelling for a moment on the fact that migraine and Raynaud's disease, although they must be regarded as two separate affections, are not infrequently encountered in the same individual. I have selected the following as a striking example:—

A married woman, aged 31, stated that for several months she had been subject to attacks in which her fingers would go white and cold and remain so for about an hour. Usually two fingers were involved and both hands were symmetrically and simultaneously affected. The attacks were especially brought on by cold.

Two months before I saw her, she had her first attack of migraine, after bathing in the sea. She first noticed a haze in the left half of her visual fields followed by the appearance of serpentine figures, left hemianopia, and numbness of the lips and hands. These sensations from start to finish lasted three-quarters of an hour and were followed by a very severe bi-frontal headache, which continued for four hours and culminated in vomiting, after which she felt perfectly well again, being able to go to a concert two hours later.

Subsequently she had experienced three further attacks. Two were repetitions of the first.

In the third the "top half of things went dim," she experienced nausea, numbness of the left arm, of both lips and both sides of the tongue, and also in the left leg. Her "inside seemed to quiver" and she had much flatulence. This was followed by a terrific headache. Next morning it was gone but she felt "woolly" all day.

There were no signs of any organic disease nor have any developed since, though she has had further attacks of migraine. There was no family history in this case, but she herself had been subject to severe bilious attacks in her childhood.

Instances have also been recorded by Osler and others of transient disturbance of vision and sensation occurring at rare intervals in the course of long-standing cases of Raynaud's disease.

Proceeding with the hypothesis of vascular spasm, can it explain the headache? The suggestion originally made by Du Bois Reymond [5], himself a sufferer, was that the arteries during an attack of migraine are in a state of tetanic contraction which is itself painful. "The latter view," he says, "takes for granted that the tonic contraction of the smooth muscular fibres is not less painful than that of the cross-striped fibre, as in cramp of the legs, tetanus from electrical tetanization and the like. The pains of labour and of colic justify us in making this concession."

I find some difficulties in the way of accepting this explanation. If tonic spasm in the arteries is at the same time the cause of the visual and sensory disturbance and of the headache, how is it that the visual or sensory disturbance as a rule precedes the headache, and that either may occur, in fully developed form, independently?

It would be more satisfactory to suppose that we have an analogy with the blue form of Raynaud's disease, and that as in that disease local asphyxia may lead to local œdema. This would occur more readily in the brain owing to its loose texture than in the fingers. This hypothesis would better account for the common sequence of events, and would, I think, explain the headache. We know from our experience of cerebral contusions that a local swelling upon the surface of the brain may be quite small and yet, provided that it is of sudden development, give rise to very severe headache. A small cerebral contusion may result in severe pains; a meningeal tumour ten times the size may never have caused a headache, presumably because the slow development of the tumour allows room for moulding and displacement, so that the tension upon the meninges is evenly distributed throughout the whole envelope and does not reach the pain threshold at any one spot.

I have one further observation to put forward in favour of this hypothesis, and that is the occurrence in some cases of true migraine, during an attack, of subarachnoid hæmorrhage. I have met with two examples of this condition, one of which I have published [15]. Goldflam [9] has particularly drawn attention to this point and has recorded five cases of the association. It is at any rate conceivable that local asphyxia and œdema might lead to local hæmorrhage and rupture into the subarachnoid space.

Finally, it is perhaps worth observing that the theory of vascular spasm would explain the occasional occurrence of monocular blindness, the spasm being presumed in such instances to affect the ophthalmic or retinal vessels. There are on record observations with the ophthalmoscope of such spasm during an attack. Higgens, quoted by Ormond [12], stated—

"that he once had a patient whom he examined on several occasions during temporary attacks of monocular blindness associated with migraine, in which he could see the retinal arteries reduced to mere threads while the veins were full and distended; by watching the fundus he could tell when vision was returning by noticing the enlargement of the arteries."

Unfortunately the data upon which the case was diagnosed as migraine are not recorded.

If the attacks are due to paroxysmal constriction of the vessels, through what mechanism is this produced? The prevailing view upon the Continent is that it is due to over-action of the cervical sympathetic. But if we accept the work of Howard Florey, published in a recent number of *Brain* [7], this cannot be true. From direct microscopic examination of the living cortex of the cat, rabbit and monkey he satisfied himself that neither stimulation of the cervical sympathetic trunk, nor of the vasoconstrictor centre in the medulla, produced any effect in the cortical vessels. The same negative results were obtained from the local application of adrenalin. Florey found, however, that local constriction of arteries and arterioles could be produced readily by the application of mechanical, electrical or thermal stimuli. Experimenting with a few chemical substances he found that a 5 per cent. solution of barium chloride caused marked constriction, and a solution made by shaking amyl nitrite with water caused marked dilatation. The other substances tested were inactive.

It would appear, then, that if vascular constriction is the cause of migraine, it must result from a purely local disturbance of function in the vessel walls. Possibly the same is true of Raynaud's disease [3] [17], for which a nervous origin has not been proved. Such local constriction might be due to some chemical substance in the circulation, or to functional defect in the muscle itself—for which we have as an analogy in the case of striped muscle, the myotonias. In any case we must assume an inherited or acquired local susceptibility.

I fear that in this part of my remarks speculation has outrun the facts, but I have dwelt upon it at some length because I feel there is enough to suggest the lines along which further investigations should be made.

Treatment.

Of the treatment of migraine there is little new to be said. Having admitted the importance of the hereditary factor in the majority of cases, we cannot speak in any strict sense of a "cure," and the liability to attacks continues, though frequently diminishing with advancing years. In almost every case, however, efficient treatment will succeed in diminishing the frequency—and often the severity—of the attacks. Of these measures regime would appear to be of the first importance. We cannot forbid worry, but we can often prescribe conditions of work, mental and physical, recreation and diet, which are of the greatest value.

Of drugs, luminal in some cases, and in others liquor trinitrini, given regularly as preventive measures, are of undoubted value. Individual patients vary in the degree of benefit which they may obtain from these two drugs, so that it is worth trying them alternately.

There are patients who claim to have discovered for themselves special precipitating causes, dietetic or otherwise.

Once the attack has begun the patient as a rule knows best from experience how to care for himself, complete mental and physical rest being the chief ingredients of his prescription. Occasionally he may be able to avert an attack by some method of his own. The most commonly successful in slight cases is a dose of aspirin and

complete rest for an hour or two. If with this, sleep is obtained, the development of the attack is often checked. Sometimes, especially if the attack begins at the end of a period of fasting, a good meal with a glass of wine will cut it short. A medical friend who has a family history of migraine and suffers from definite attacks with visual disturbance tells me he can often avert them by hanging his head downwards for a few minutes. I have advised this measure in other cases without benefit.

Of refractive errors I have already stated my opinion that they may cause recurrent headaches on their own account, but are not an important cause of migraine. The question will doubtless be debated with acrimony. I will conclude my remarks upon this point by citing in my support Mr. Foster Moore [11] who in the most recent edition of his book "Medical Ophthalmology," says: "Whilst nothing but good will come of the correction of any real error of refraction that may exist, there is, I believe, no direct causal relationship between any ocular abnormality, refractive or otherwise, and migraine."

Summary.

In summary of these remarks I would say that the evidence which I have reviewed suggests that migraine is due to a susceptibility, inherited or acquired, of the cortical vessels to spasm of their walls. There is nothing to show by what means this spasm is provoked, but mental stress in some way, at present obscure, plays an important part. Possibly the reaction is of a chemical nature and is due to the action upon the vessel walls of toxins derived from neural metabolism.

Other toxins derived from faulty metabolism of fats, sugars or proteins may also be responsible.

There is some evidence to show that the attack may be associated with an alkalosis or with an excess of cholesterin in the blood.

There would seem to be at least two lines of investigation worth pursuing. On the experimental side, further observations should be made along the path opened up by Florey—the effect of various chemical substances, e.g., cholesterin, acetone derivatives, acids and alkalis, upon the cortical vessels, either when applied directly or introduced into the blood-stream. On the clinical side, a number of cases of true migraine, coming within the definition of this paper, should be investigated by the clinician and biochemist in collaboration during the free period, before, during and after the attack. From a comparison of the results obtained by two or three teams working upon these lines we might hope to gain some positive knowledge of the part played by diet and faulty metabolism in the causation of the attacks, and so set this part of our treatment upon a firmer basis.

References.

[1] Allison, *Brit. Med. Journ.*, 1297 (i), 165. [2] *Brit. Med. Journ.*, 1926 (ii), 765. [3] Brown, *Arch. Inter. Med.*, 1925, xxxv, 56. [4] Curtis-Brown, *Brit. Med. Journ.*, 1925 (i), 155. [5] Du Bois Reymond (quoted by Liveing). [6] Fawkes, *Brit. Med. Journ.*, 1926 (ii), 1176. [7] Florey, *Brain*, 1925, xlviii, 43. [8] Fothergill (quoted by Liveing). [9] Goldflam, *Deutsch. Zeitschr. f. Nervenheilk.*, Leipz., 1923, lxxvi, 158. [10] Liveing, "On Megrim, Sick Headache and Some Allied Disorders," London, 1873. [11] Moore, R. Foster, "Medical Ophthalmology," J. A. Churchill, London, 1925. [12] Ormond, *Trans. Ophth. Soc.*, 1913, xxxiii, 138. [13] Pagniez and Nast, *Presse méd.*, 1920, April 28, 253. [14] Paulian, *Rev. Neurol.*, 1925, ii, 947. [15] Symonds, *Quart. Med. Journ.*, 1924, xviii. 93 [16] Weismann-Netter, *Comptes-rend. Soc. de Biol.*, 1925, xcii, 341. [17] Wolf, *Heart*, 1924, xi, 327. [18] *Rev. Neurol.*, 1925, ii, 854

Section of Medicine 23

Dr. HUGH THURSFIELD (President)

regretted that a medical friend (aged 50) of his could not personally be present, but he had written an account of his symptoms, which he now proposed to read, with the addition to the account of what he himself had observed in the bad attack recorded, the patient's record of which is, perhaps naturally, inadequate.

My first experience of migraine was at the age of 18 when a student. The attack occurred at about noon. It was of the same character as most of my attacks. A central clear space of an elliptical shape surrounded by an irregular luminous, in fact dazzling border beyond which was a misty area. On closing the eyes the effect was changed to a central dark spot with a very dazzling frame, outside of which was an area of rapidly revolving bodies. The sensation gradually increased in intensity to a maximum and then gradually passed completely away. These attacks usually lasted for about 10 to 20 minutes. Sometimes the right eye only was affected, sometimes both. As a rule a headache followed in about half an hour or so, but I soon found that I could prevent this by taking a phenacetin tablet directly I got the first symptoms. Not being a "sick" subject I never had any vomiting or even feeling of sickness. I have often noticed my right pupil larger than the left, and I believe, though I cannot be certain, that this was generally more marked during an attack. In my own mind I am quite sure that the attacks are most commonly the sequel of slight digestive disturbance sometimes associated with nervous exhaustion following continuous hard work especially if the latter is accompanied by mental worry. My two worst attacks (of a different character) followed a time of marked mental and physical exhaustion after very hard work. The attacks most frequently occurred in the morning between 9 and 11 but occasionally at later hours in the day. From my personal experience I generally treated my patients with an initial dose of aperient of a saline nature followed by phenacetin or potassium bromide and have usually found this treatment satisfactory in ordinary attacks.

In 1920 I had an attack of a different type. When dressing in the morning I suddenly noticed that I had slight diplopia. On looking in the glass I found that internal squint was present. Thinking that this was only an ordinary attack of migraine I expected the usual visual effect. Instead of that the diplopia continued and later the vision became so obscured that I had to rest. There was also some mental confusion as, although I thought I was perfectly clear and indeed said so, yet I learnt afterward that I made several silly remarks and feeling rather sleepy I agreed to go back to bed. Drowsiness supervened and although I awoke when anyone came into the room yet I found the diplopia still present. Headache was present to a large degree, but I was perfectly certain that the whole thing was of the migrainous type and that it would pass off in a short time and assured my friends of this. Early in the afternoon this had occurred and I found that I was practically all right again, but being a little afraid that the diplopia might return I kept quiet for the rest of the day. The next day I was up and about and indeed surprised some of my friends who had been told that I had had a "stroke."

[*Note by Dr. Thursfield.*—This does not fully describe the real sequence of events, as ascertained at the time. The attack began in the usual way with a diplopia, but instead of passing off in a short time this was succeeded by complete blindness, some mental confusion, a partial right hemiplegia, especially noticeable in the right side of the face. He was carried to bed, and when seen by me three hours later still had an obvious weakness of the right facial muscles; and also some noticeable mental confusion, and inability to express himself properly. Of this he was aware himself, and explained that "he couldn't remember" the name or the word that he wanted. He also had a slight definite weakness of the right external rectus muscle. A night's rest

completely restored him, and he had perfectly recovered from all his symptoms next morning.]

About two years later I had another attack of a different kind. While giving an anæsthetic I suddenly became aware that I seemed to have some loss of power in my right arm and had to exercise considerable mental power to lift it up and move it about. It felt numbed and unnatural. At the same time I found that when I attempted to speak I was inclined to mumble and had difficulty in articulating clearly. There was also a tendency to laugh, due, apparently, to a twitching of the corners of my mouth, especially the left. I said to the operator "I have a slight attack of migraine, so do not worry if I seem funny as it will soon pass off." I was able to continue anæsthetics of two or three more cases and then went on my rounds. I had, however, to explain to several people what was the matter as the tendency of my mouth to twitch and also to laugh continued for some time. During the afternoon it gradually wore off. There was a slight drowsy tendency, but not enough to interfere with my work which I succeeded in getting through.

As far as I can remember the numbed sensation also was present in my right leg, but to a less extent, causing me to be extra careful in moving about. Mentally I was quite clear and perfectly certain that it was another attack of the same kind as before. The attack interested me in that it seemed to be of the nature of a functional hemiplegia. It gradually increased to a certain severity and then passed off in a similar way to all my other attacks.

Dr. A. F. Hurst.

I agree with Dr. Symonds that migraine depends primarily upon a constitutional and often inherited irritability of some part of the central nervous system. But I believe that the most common and important factor which increases this irritability is eye-strain. In my experience of a large number of both typical and atypical cases improvement, often amounting to the complete cessation of attacks, almost invariably follows the accurate correction of errors of refraction and of disturbances in the balance of the external ocular muscles. Unfortunately very few oculists take sufficient trouble in cases of the kind, but the few who have been convinced of the need of extreme accuracy in order to relieve eye-strain, have been rewarded by obtaining most satisfactory results. In recent years I have found luminal a very useful drug in reducing the excitability of the migraine "centre," but it is not essential, and it can generally be discontinued as soon as the ocular errors have been sufficiently corrected. There is no doubt also that fatigue and toxæmias of every kind increase the liability to attacks, as exhaustion of the nervous system is accompanied by increased irritability.

My colleagues and I have investigated a considerable number of cases by biochemical and radiological methods, but we have never found any constant abnormality. Difficulty in the digestion and metabolism of meat, fat and carbohydrates, hepatic and renal insufficiency, dilatation of the stomach, intestinal stasis, and toxic idiopathies, have all been regarded at different times as the cause of migraine, but in no case were any of these abnormalities discovered. There is no doubt that many women are specially liable to attacks during menstruation, as John Fordyce pointed out 150 years ago, and attacks may cease temporarily during pregnancy and permanently at the menopause. This must clearly be due to some endocrine activity which renders the storm-centre particularly sensitive during menstruation, but with this exception there is no evidence that migraine is in any way associated with disorders of internal secretion.

Radiological investigations have shown that during an attack gastric peristalsis

ceases and a condition of atony occurs, but this is part of the migraine and not a cause. The starvation which accompanies an attack and the loss of fluid following severe bouts of vomiting also give rise to various biochemical changes, which have been wrongly regarded as evidence of a toxic origin.

Finally, I should like to urge those whose experience has led them to regard eye-strain as of minor importance to choose another oculist, or, if necessary, a series of oculists, to examine their migrainous patients. It is necessary to add that the patients must conscientiously wear the spectacles they are given all the time and must not let vanity interfere with their cure.

I have known an attack of typical migraine with hemianopia, fortification phenomena, hemicrania and vomiting occur after an interval of four years in a young woman whose very frequent and severe attacks had been completely arrested by proper spectacles after prolonged drug treatment and rest had failed. She had given up using them for over a year, when a period of physical fatigue and worry brought on the relapse. She at once returned to the discarded spectacles, and no further attacks have occurred in the four years which have since elapsed.

Dr. E. Arnold Carmichael.

What is the opinion of Dr. Symonds as to the hypothesis which bases the symptomatology of migraine upon a transient unilateral or bilateral hydrocephalus? There are several points in favour of such a hypothesis.

(1) In cases of cerebral tumour, hydrocephalus of the opposite cerebral hemisphere frequently occurs with a resulting large pupil upon the same side. I believe, in persons suffering from hemicranial headaches of migraine, that the ipsilateral eye is dilated. This is a point suggestive of a unilateral hydrocephalus being the underlying cause.

(2) The symptomatology of migraine may be likened to a "slow" epileptiform seizure—the lights before the eyes being positive phenomena and the partial paresis a negative phenomenon. In hydrocephalus, headaches, vomiting and fits followed by varying degrees of transient paresis occur.

(3) The fact that the calcarine fissure causes an elevation on the inner surface of the posterior horn of the ventricle renders this region liable to the earliest effect of hydrocephalus. This would be consistent with the hemianopic visual disturbances met with in migraine.

(4) Spitzner has published the records of several cases of migraine where at post-mortem a degree of hydrocephalus was found.

Dr. J. Kingston Barton

said he was surprised by Dr. Hurst's statement that all, if not nearly all, cases of migraine were cured if only the patients succeeded in finding an ophthalmic surgeon who was competent enough to find out the error of refraction which was the real underlying cause of all these cases. In his (Dr. Barton's) experience a certain number of people with most troublesome head and eye symptoms had been cured by proper spectacles being prescribed. But in most of these eye cases the patients rarely exhibited many of the stigmata or the syndrome signs that made up true migraine. Dr. Symonds had not mentioned one very important symptom which he (Dr. Barton) believed to be a crucial test of a case being true migraine, namely, the copious polyuria

that the patient developed during the height of the attack. So much so that during the period of headache, the sickness and polyuria, the throbbing heart, and the many contracted small blood-vessels (similar to those which Dr. Symonds held might be present within the cranium), almost supplied a small clinical picture of a case of contracted cirrhotic kidney. This would point to some substance requiring elimination being present in the blood, and making the heart and blood-vessels behave in a way similar to what happened in a case of actual ablation of kidney substance. If it were admitted that vascular spasm within the brain was probably the anatomical feature of migraine then one would look for something that acted through the medulla of the adrenal organs and the vasomotor system generally. Dr. Hurst's contribution to the subject of the motor paralysis of the walls of stomach was a point worth following up. It should be remembered how frequently those subject to migraine suffered with that positive exhibition of vasomotor disturbance, namely, "dead man's fingers."

It was most important that the modern medical units in hospital medical schools should use all their resources to investigate the many sidelights brought to bear on recognized cases. Young house surgeons or physicians, or probationer nurses, were the most likely persons to come across such patients on the spot.

A well-known symptom in migraine was the extraordinary feeling of *bien être* or well-being just before an attack was coming on. That was the time for biochemical investigations into the blood uric acid, the blood reaction itself, blood urea, blood cholesterol, creatinin, blood-pressures, the urine excreted hour by hour, also for ophthalmoscopic examinations, examinations of heart sounds, etc. The interval of *bien être* was generally well marked towards bed-time; the storm usually broke in the early morning.

It had been stated that many of these sufferers ceased to have attacks after 50 years of age. In his (Dr. Barton's) experience most women lost their migraine after the onset of the climacteric, but when they continued to suffer from bad sick headaches after 55 he invariably found that they had not only inherited the gouty phenomena but had continued to eat and drink much too freely, and were only cured by strict attention to diet and a yearly course of treatment at Aix-les-Bains, or Carlsbad. Men seemed to suffer less as they grew older but only because they had learned to avoid excess of meat and to be very chary of taking any alcohol. In fact, large numbers of sufferers cured themselves by a severe limitation of meat diet in daily life (especially in the case of women), and practically complete abstention from alcohol. He thought the older authors were right when they put migraine, asthma, skin affections, etc., in a group under inherited gout.

Mr. Herbert W. Nott

briefly discussed the results obtained by permanganate treatment. It had been stated in the course of the discussion that the most probable cause of migraine, and other allied conditions, was a floating toxin in the blood, the ill-effects of which might often be controlled by means of a treatment which was found to exercise a rectifying effect upon metabolism, and in cases in which other remedies had failed.

Dr. J. A. Ryle

said there was one pertinent question which could be put in regard to migraine, in common with the other intermittent, "explosive," disorders (namely, asthma, epilepsy, and gout), and this was: "Why were they all incurable?" For it must be

admitted that, however much we might be able to alleviate the symptoms or diminish the frequency of the attacks in these disorders, no cure in the strict sense could be achieved. Surely the answer to this question was that the disorders depended upon an inherited predisposition and that this predisposition, being, in the biological sense, a true variation and as such transmissible, was from the physician's point of view the ineradicable factor. Dr. Symonds had referred to the frequency with which migrainous patients gave strong family histories. It was not improbable that in *every* case the hereditary factor was present. "Skipped generations" could be reasonably accounted for by students of heredity and were, perhaps, more common in conditions in which transmission took place more evidently through one sex, as was the case with migraine. The discontent of the laity with the achievements of the medical profession in regard to these disorders was really unjust, for in effect they were expecting to be relieved of something which was a part of the general constitution with which they had been endowed by their ancestors.

Dr. Ryle also reminded the meeting of an observation of Lauder Brunton, supporting the view that arterial spasm was a factor in causing the pain of migraine. Sir Lauder Brunton was able to demonstrate in his own case that his temporal arteries were hard and contracted during the period of the headaches, although soft and relaxed at other times.

Dr. Agnes Savill

said she believed migraine was often primarily of toxic origin, the error of refraction so frequently present being only a contributory or exciting cause. She referred to a family in which several members developed migraine when their vitality was low; the attacks came on, with the regularity of a clock, day after day at the same hour. In one case the migraine was arrested by a course of Carlsbad salts; in another sod. bicarb. with sod. salicyl. always cured the condition. Refraction errors were present, but though the migraine had been arrested by the methods mentioned, the glasses had not been altered.

Mr. Frank Coke

mentioned a case in which violent attacks of migraine had been associated with asthma due to feather sensitiveness, and another in which a sufferer from migraine, sensitive to pollen, was free from migraine during the time that he was suffering from hay fever.

Dr. W. R. Reynell.

The problem of migraine is merely a part of the larger problem of the paroxysmal neuroses, the epilepsies, asthma and the rest. It is almost certain that in all of these there is some inherited constitutional defect. The analogy between epilepsy and migraine is striking—the relatively large heredity and constitutional factors in both with similar groups of exciting causes—psychogenic, metabolic, infective and toxic. In both the symptoms are due to disturbances in the central nervous system, and in both the typical attack may be replaced by what is called a "psychic equivalent," in which the symptoms are those of a paroxysmal psychosis. Migrainous patients often give a history of night terrors or cyclical vomiting in childhood and

in cases of epilepsy there may be a history of enuresis or somnambulism in early life. When the problem of epilepsy is solved we shall know more about migraine.

Dr. F. W. Collingwood.

I have been a subject of migraine for the whole of my life, and with two dioptres of hypermetropia. I have never had the slightest trouble after using my eyes in reading for prolonged periods. But the occasions on which the worst attacks have ensued have been after debates in which controversial questions have arisen, especially those of a political type.

It appears that in my case the state is hereditary, several of my ancestors and relatives having suffered from migraine.

Section of Ophthalmology.

President—Mr. Ernest Clarke, C.V.O., F.R.C.S.

CLINICAL MEETING.

Held at the Central London Ophthalmic Hospital, Judd Street, December 10, 1926.

Peculiar Opacity of Cornea.

By Leslie Paton, F.R.C.S.

I saw this patient a week ago. He had then been under treatment for over two years as a case of subacute glaucoma. I think the diagnosis was based on the occurrence of haloes. I could not myself find evidence of glaucoma except for these haloes. The tension was somewhat subnormal, the anterior chambers were fairly deep, there was no trace of venous pulsation, nor of cupping of the disc, and no real evidence of any condition pointing to glaucoma. The vision was good, considering the amount of opacity in the cornea, namely, $\frac{6}{9}$ with correction in the right eye, $\frac{6}{60}$ in the left eye, but he has not used the left eye for a long time. He has a persistent pupillary membrane in the left eye. The explanation of the haloes lies in the deep haze in the cornea, which is the result of old keratitis profunda.

The main reason for bringing the case is to obtain the opinions of the slit-lamp experts on it. Most of us are at present only amateurs in the use of the slit-lamp. I would like opinions on the curious appearance of haze in the left eye, not exactly on Descemet's membrane, but in the very deep layers of the substantia propria. There are curious little rosettes of spindles, the spindles having a double contour. They are not, I think, crystalline, and the position is against them being folds in Descemet's membrane. It is these rosettes of spindles forming a mosaic in the very deep layers of the cornea which perplex me, and I would like to know if any Members have seen anything like it before. I have not.

Recurrent Intra-Ocular Hæmorrhage in a Young Male.

By H. M. Gilmour, L.R.C.P.Lond., M.R.C.S.Eng. (Ipswich).

Patient, a farm labourer, aged 23, who saw me on September 23, complaining that vision had failed three weeks previously. Right vision = $\frac{6}{12}$, left vision = $\frac{6}{6}$.

Right eye shows recent blood-clot in the vitreous and a retinal detachment below. Left eye has a mass of fibrous tissue in the vitreous, presumably the consequence of hæmorrhage at some former time, no retinal detachment can be demonstrated. In both eyes transillumination is good.

We excluded syphilis, diabetes, nephritis; all the organs were examined and found healthy. No von Pirquet test has been made. Seeking a cause I considered that the most profitable line of inquiry would be along the assumption that a mild infection of the blood was present; some support for this supposition was provided by the fact that the patient during the first three weeks had an intermittent elevation of temperature. The teeth were X-rayed but nothing was shown to be wrong with

44 Mayou: *Industrial Toxic Amblyopia*

their roots, nor, indeed, did we find any infective focus. The condition is now practically identical with the state in which I first saw him eleven weeks ago.

Discussion.—Mr. GRAY CLEGG said he considered that in this patient's left eye there was a detachment of the retina below. Vessels could be seen, and, in addition, vitreous opacities. He wondered whether an altered coagulation point in the patient's blood was the explanation of the hæmorrhages.

Mr. A. L. WHITEHEAD said that some time ago he recorded a case before the Section which was somewhat similar to that shown by Mr. Gilmour. It was that of a young man who had, apparently, the ordinary type of vitreous hæmorrhages. Ultimately, six to twelve months after the onset, he developed a typical tuberculous iritis in one eye. Tuberculin treatment was begun, and the condition began to improve at once. At his worst he had mere perception of light in one eye, and with the other eye he could count fingers a yard away; but after the tuberculin treatment vision in one eye was $\frac{6}{9}$, in the other $\frac{6}{6}$. The iritis cleared up with the exception of some strands and patches of retinitis proliferans, which, however, did not interfere with his macular vision. He (Mr. Whitehead) was therefore encouraged to recommend a trial of tuberculin in Mr. Gilmour's case.

Industrial Toxic Amblyopia.

By M. S. MAYOU, F.R.C.S.

THIS case is a patient of Mr. Munro, of Ipswich. It has an important aspect from an industrial point of view. He is a farm labourer. His discs are very pale, the vessels are of good size, and he has a large central scotoma, with slightly contracted fields. Vision = H.M. each eye. His occupation is that of spraying fruit trees with a mixture of arsenic, nicotine and lead, and since he started this work his sight has gradually deteriorated; now it only amounts to perception of hand movements. I regard this as a case of nicotine poisoning brought on by the use of the nicotine and other poisons used in the spraying. It appears that while the spraying is in progress the man is in a complete mist of the mixture.

When he first presented himself it was thought the only other possibility was pituitary enlargement. He has been under Dr. Martin, who examined him, with X-rays and otherwise, but can find no alteration of sugar tolerance or any other change indicative of pituitary disease. I made inquiries at the Ministry of Agriculture as to whether similar cases had been reported to them: their reply was in the negative.

Dr. PURDON MARTIN said this patient was first sent to him, as a case of optic atrophy, to be medically examined. He had the history that the patient had been using a nicotine preparation, but the visual fields had not been taken, and several other possibilities were considered. The patient had not the general appearance of a case of pituitary tumour, but the discs were more pale in one half than in the other, and there was the peculiar appearance of skin which patients with pituitary disease often had; it was a soft white skin; there was also a marked deficiency of hair, and the patient had never required to shave more than once a week. For those reasons it seemed that a pituitary condition might at least be a factor. Also, the man had a definitely oxycephalic skull, though he (Dr. Martin) did not know whether it was pathologically so. Oxycephaly was therefore another possible factor. Since then the case had been investigated at the National Hospital, Queen Square, by X-rays and otherwise. The radiograms did not reveal any abnormality in the pituitary fossa, nor was there any disturbance of the blood-sugar curve; the fields of vision showed that the visual disturbance was due to a large central scotoma, and was not of the pituitary type; there were no indications of syphilis. He agreed that the atrophy in this case was probably toxic.

Section of Ophthalmology 45

A Slit Lamp.

Shown by M. S. Mayou, F.R.C.S.

THE instrument exhibited is an improvement on that described in the *British Journal of Ophthalmology* in March, 1926.

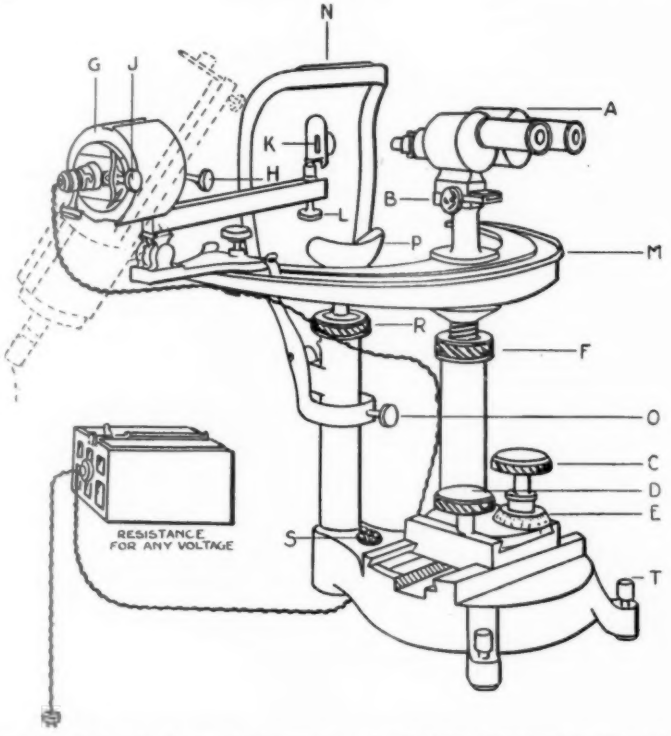
Its advantages are the following:—

(1) When once the lamp is in focus no further adjustments need be made for the examination of any number of patients, and consequently the average examination does not require more than about one minute.

(2) The lamp and microscope move in unison so that one adjustment does for both focusing the image and directing the light.

(3) The instrument is portable and can be used on any small table with a glass top, and when not in use can be put out of the way.

(4) The slit can more accurately be focused on any portion of the anterior part of eye.



A.—Zeiss or any other make of corneal microscope.
B.—Focus adjustment for A.
C.—Focus adjustment for microscope and lamp.
D.—Lateral adjustment for microscope and lamp.
E.—Vernier drum for micrometer measurements.
F.—Adjustment collar for height of microscope and light.
G.—Air-cooled lamp chamber.
H.—Adjustment for width of slit.
J.—Centring adjustment for filament.
K.—Condensing lens.
L.—Height and focus adjustment for condensing lens.
M.—Slotted arc on which lamp travels.
N.—Adjustable head rest.
O.—Adjustment for head rest.
P.—Adjustable chin cup.
R.—Adjustment for chin cup.
S.—Switch for light.
T.—Screw adjustment for raising on domed feet.

(For the loan of the above block the author is indebted to Messrs. Hamblin, Ltd..).

46 Neame: *Melanoma of Conjunctiva;* Gann: *Pigmentation of Fundi*

Familial Macular Degeneration.

By HUMPHREY NEAME, F.R.C.S.

PATIENT, a woman, M. S., aged 40, who says she has always had defective vision. I managed to see her brother, F. B., and examine him, and I found he had vision to the extent of $\frac{2}{12}$ only. I could not get him to read $\frac{6}{60}$ with correction; he had a high error of refraction, a mixed astigmatism of 3·5 dioptres. He frequently drives a motor between Kingston and London, and, up to the present, he has not had an accident. The brother's macular degeneration is less than the sister's. He simply has a delicately marked pigmented ring at the centre of the macula. The sister has a more marked change, with pale patches and streaks at the macular region, and slight pigment around them. There is a third member of the family, another brother, whom I have not been able to see. From the accounts of the brother and sister, evidently he also has defective vision, but I am not able to verify the condition of his fundus. The woman you have seen has vision $\frac{6}{36}$ in each eye.

Melanoma of Conjunctiva.

By HUMPHREY NEAME, F.R.C.S.

PATIENT, a male, aged 60. Occupation, brass finisher.

History.—A brown spot had been noticed on the white of the left eye since an injury from sulphuric acid at the age of about fourteen years. The patient came to hospital for examination for glasses because of headaches with near work. Right vision $\frac{6}{24}$ + 2·5 D. = $\frac{6}{6}$. Left vision $\frac{6}{24}$ + 2·5 D. = $\frac{6}{6}$.

Present condition.—A dark brown slightly raised patch in the conjunctiva close to the limbus at 10 to 11 o'clock. It is movable over the sclerotic except where tethered at the limbus. Coloured drawing made October 9, 1926.

This is an interesting example of pigmented tumour, of which I had a drawing made, in order that I may see whether there is any increase in size. If so, I shall excise the growth as freely as possible, from the surface of the eyeball.

Congenital Pigmentation of the Fundi.

By J. HURNDALL GANN, L.R.C.P.Lond., M.R.C.S.Eng. (Introduced by Mr. HUMPHREY NEAME.)

PATIENT, a girl, aged 8.

History.—Measles three years ago. Left eye became red and was afterwards noticed by parents to turn in. Out-patient Royal London Ophthalmic Hospital, under care of Mr. Lang in November, 1923 and August, 1925. Lenses prescribed for constant wear.

July, 1926.—Left convergent comitant squint. Now wearing.—RIGHT: $\frac{6}{36}$, + 3·00 sph. + 2·00 cyl., 90° = $\frac{6}{9}$. LEFT: $\frac{2}{60}$, + 4·00 sph. + 2·00 cyl., 90° = $\frac{6}{60}$.

Numerous shaded black patches of irregular size and shape are scattered over both fundi. The largest are towards the periphery, and are all deep to the retinal vessels.

Mr. HUMPHREY NEAME said that he got into touch with Mr. Lang about this case. Mr. Lang said that the patient had been shown a year ago at a meeting of the Section, at the same time as two other cases, one a case of marked pigmentation in a congenital syphilitic choroiditis, and the other a case of retinitis pigmentosa. When they were placed side by side the difference was apparent. In the present case there were discrete circumscribed patches, which had been described as sarcinoid groups. Some of the marks resembled the footprint of an animal.

Associated Movement of Jaw and Lid.

By F. A. JULER, F.R.C.S.

THE case of this patient, a girl, aged 5, illustrates jaw-winking movements, i.e., associated movements of the jaw and upper lid, a condition which, I think, was first described by Marcus Gunn many years ago. I have seen only one or two other cases. It affects one lid only, in this case the left upper lid. At rest the lid droops nearly completely, but when the child opens her mouth, as in chewing, the upper lid goes up into its proper position. Presumably there must be some abnormal connexion between the motor root of the fifth nerve supplying the external pterygoid and the nerve to the levator muscle, the upper division of the third.

Mr. GRAY CLEGG said that this case reminded him of that of a boy, in whom, when he was eating, especially sweet things, epiphora began to appear.

MISS IDA C. MANN, F.R.C.S., showed the following cases: (1) Unusual Case of Retinitis Proliferans. (2) Result of Plastic Operation for Rodent Ulcer of Lower Lid. (3) Arterio-Sclerotic Retinitis.

? Choroidal Exudate.

By IDA C. MANN, F.R.C.S.

I HAVE shown this case because I would like to hear opinions as to its nature. The patient has no albuminuria, nor are the retinal vessels obviously sclerotic though the blood-pressure is 230 mm. Hg. The fundi show glistening white dots, particularly grouped in the temporal part of each. They are deep to the retinal vessels, which run over them unchanged. There are also larger pale areas which would seem to lie deep to the retina in the choroid. I am uncertain whether to class the case as one of choroidal exudate or as an atypical form of retinitis circinata.

Mr. ERNEST CLARKE (President) said this condition was much more like a choroidal exudate than retinitis circinata; it certainly had no resemblance to a typical case of the latter.

Case of (?) Blood-staining of the Cornea.

By H. P. GIBB, F.R.C.S.

Mr. ERNEST CLARKE (President) reminded the Members that the stain (hæmatoidin) was absorbed from the periphery to the centre, and he said that this case was shown at too late a stage to exhibit the extraordinary resemblance the condition had to the lens dislocated forwards and lying at the back of the cornea, which was the appearance of an early typical case. Thirty years ago Mr. Treacher Collins read a paper on this subject before the Ophthalmological Society,[1] and twenty-three years ago he (the speaker) showed a typical case of it.[2]

1 *Trans. Ophth. Soc.*, U.K. (1894-95), 1895, xv, 69.
2 *Ibid.* (1902-3), 1903, xxiii, 322.

48 Cunningham: *Cases of Oxycephaly, etc.*

Cases of (1) Oxycephaly; (2) Discoid Cataract; (3) Coronary Cataract; (4) Cyst of Iris; (5) Perivasculitis; (6) Evulsion of Optic Nerve; (7) Vascular Disease; (8) Glycosuric Retinitis.

By J. F. CUNNINGHAM, F.R.C.S.

I HAVE skiagrams of the case of oxycephaly. They show the flattened orbit. The case was seen by Dr. Adie, who pointed out the typical changes in the hands, etc.

In the case of discoid cataract I propose to do a central iridectomy, or even an iridotomy.

With regard to the case of cyst of the iris, the patient (a boy) had measles when he was young. There is a whitish mass behind the iris and it is possible that the condition may be secondary to this. The eye is a blind one, and it is questionable whether the parents will agree to it being removed.

Section of Orthopædics.

President—Dr. W. T. Gordon Pugh.

DISCUSSION ON THE TREATMENT OF PES CAVUS.

Mr. A. Rocyn Jones.

Pes cavus, or hollow foot, is the name given to a foot with an unusually high longitudinal arch. There is, however, great variation in the relative heights of the arch in quite normal feet, and often a hollow foot of mild degree causes no trouble whatever. A pure cavus deformity is by no means one of the commoner deformities of the foot; it is, however, frequently associated with some other deformity, like varus or equinus, but I shall deal mainly with true or essential cavus.

Ætiology.

The ætiology is often not very clear, chiefly owing to the fact that the disability does not become manifest until long after the exciting cause has disappeared, or has been forgotten. There are, however, distinct contributory causes, and these may be conveniently classified into three main groups, postural, paralytic and traumatic, and subdivided as follows:—

I.—*Postural*: (*a*) Faulty footwear. The wearing of high-heeled short shoes. (*b*) Habit gait. Compensatory for a short limb.

II.—*Paralytic*: (*a*) Acute specific fevers. Paresis, or slight paralysis following rheumatism, scarlet fever, measles, diphtheria, etc. (*b*) Infantile paralysis. (*c*) Chronic nervous disease, e.g., Friedreich's ataxia and progressive muscular atrophy. (*d*) Spinal cord lesions (congenital). Usually associated with skeletal malformations, e.g., spinal bifida occulta and wedge-vertebræ. (*e*) External popliteal nerve injury.

III.—*Traumatic*: (*a*) Foot injury. (*b*) Racial custom, e.g., Chinese foot.

Acute specific fevers and other acute illnesses are perhaps responsible for the majority of hollow feet. Infantile paralysis comes next, but this disease is not so productive of essential cavus as that of a mixed cavus deformity, such as cavo-varus or calcaneo-cavus. Friedreich's ataxia and peroneal muscular atrophy are responsible for some of the most severe types of pes cavus; the convexity of the long arch is often greatly increased. An interesting class is that associated with [1] spinal malformations; some of these have no clinical evidence of spinal abnormality; the defect is only revealed by radiographs. There is undoubtedly an accompanying spinal cord lesion of some kind. The only remaining class that need be mentioned is that due to faulty footwear. This is not so productive of painful hollow feet as would appear. A good number of females wear high-heeled short shoes, yet comparatively few require operative interference for pes cavus.

Mechanism of Pes Cavus.

The maintenance of the long arch of the foot depends primarily upon the tone of the muscles. Those in the sole, including the abductors, short and long flexors of the toes and tibialis posticus increase the height of the arch by approximating the

42 Rocyn Jones: *The Treatment of Pes Cavus*

toes to the heel; whereas the muscles attached to the dorsum of the foot by opposing each other when pulling upward in front of and behind the ankle, and aided by the downward thrust of the tibia at the summit of the convexity, tend to flatten the arch (see fig. 1). Paralysis of the tendo Achillis will cause the heel

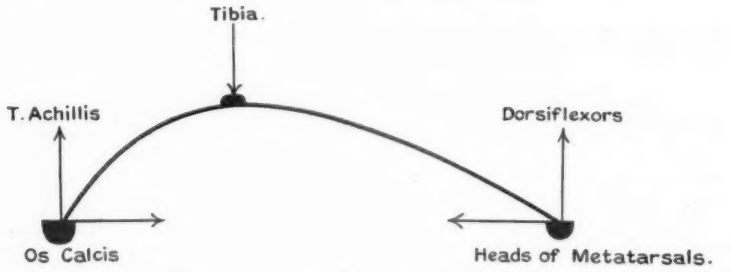


FIG. 1.—Diagram to show the direction of pull of the muscles acting upon the long arch of the foot.

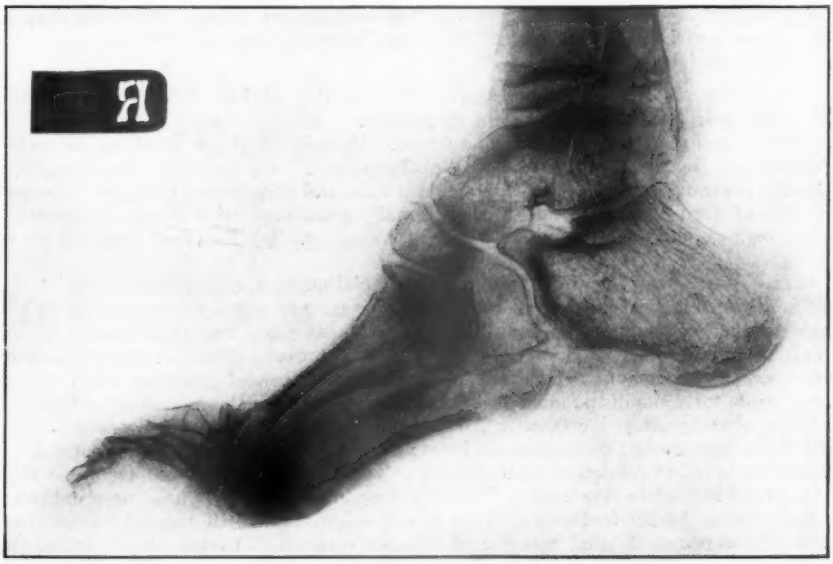


FIG. 2.—Radiograph showing the site of tarsectomy which in this case is limited to the cuneiform bones. The correction clinically was complete. See also fig. 4.

to be drawn towards the toes by the plantar muscles, thus producing a cavus deformity, and the opposing dorsiflexors will tilt the foot upwards, producing a calcaneus heel. When, however, these last muscles are paralysed an equino-cavus deformity is produced.

In the large group of cases following an acute specific fever or other illness, a passing paralysis, or paresis of either the tibialis anticus, or extensor longus digitorum, would explain the development of pes cavus. A little awkwardness in

FIG. 3.—G. W. After correction for severe double pes cavus due to progressive muscular atrophy. Casts of his feet before operation are on his right. Tarsectomy was done on the left side only, being the worse foot.

walking, due to slight foot-drop, is sometimes noticed after recovery from the illness and the weakness lasts just long enough to enable the plantar muscles and fascia to shorten. The paralysis disappears and the gait improves, but later, during adoles-

cence, with quickened growth of the foot, the contracted plantar fascia acts like a taut tie-band, pulling the pillars of the arch towards each other, and impeding its expansion antero-posteriorly; the convexity of the arch is thus increased and pes cavus is produced with pain on walking.

Another explanation of these cases is that put forward by Duchenne on the analogy of "main en griffe" produced by ulnar paralysis. He attributed pes cavus and the associated clawing of the toes to paralysis of the interossei and lumbrical muscles, thus allowing the long and short extensors of the toes to overact by hyperextending the first phalanges and flexing the two distal phalanges. The metatarsal heads are depressed, the arch is foreshortened and the plantar fascia contracts.

Recently another suggestion has been made by C. A. Parker [2], of Chicago, who believes that the deformity is due to a perversion of the normal reciprocal action between the flexors and extensors of the toes.

In the frankly paralytic case the same mechanism is at work, but here the paralysis persists, allowing the plantar muscles and fascia to contract even more, with resulting grosser structural changes.

Clinical Types.

Regarded clinically pes cavus may assume any degree of severity, from that of a foot showing little structural alteration to that of one presenting a gross bony deformity. But speaking broadly there are two main clinical varieties and these were separated many years ago by F. R. Fisher [3], one being called *talipes arcuatus*, the other *talipes plantaris*. The former is that type of cavus in which the heel and ball of the foot are in the same horizontal plane; but if the ball assumes a lower level than the heel then the condition is called talipes plantaris. This distinction is best brought out by passively dorsiflexing the foot. It is a useful working classification, for by the use of these terms the precise type of foot we are dealing with is readily conveyed without further description. Talipes arcuatus stands for the less severe degrees of pes cavus, whereas talipes plantaris represents a further developed stage of the deformity with greater retraction of the toes and the typical claw-foot. But although the anterior part of the foot may be dropped considerably and an appearance of talipes equinus simulated, yet there is often little or no shortening of the tendo Achillis.

The patient suffering from pes cavus usually seeks advice because of continued fatigue or actual pain in the feet when walking. This is due to the limited treading surface of the sole and to excessive pressure on the heads of the metatarsals; the frequent presence of callosities at the ball of the foot bear witness to this undue metatarsal thrust.

Treatment.

(1) *Pes Cavus of Slight Degree.*

In cases of slight pes cavus and a tendency to contraction of the toes the mother is taught to manipulate the toes and encourage the child to flex and extend them. At night a rectangular slotted metal splint is worn, with tapes binding the toes to it. The boots must be sufficiently long and roomy enough to allow proper play for the toes. Rarely is any cutting required; at most a simple tenotomy of the plantar fascia and stretching the sole is all that need be done.

(2) *Pes Cavus of Moderate Degree.*

With older children and those approaching adolescence, it may be enough to teach them toe-flexion and extension exercises and increase the treading surface of the foot by prescribing a modified shoe in which a steel arch is introduced extending

FIG. 4.—C. D., after operation, showing feet corrected; casts of his feet before operation are beside him. Tarsectomy was performed on the right side, Steindler's operation only on the left. The radiograph of his right foot after operation is seen in fig. 2. There was no evidence of nervous disease present.

along its waist from heel to tread; a low plate with a lift behind the metatarsal heads, introduced into an ordinary boot, sometimes takes the place of the special shoe. If painful metatarsal callosities are present a transverse leather bar is added

to the sole of the boot behind the position of the corns. If these procedures do not give sufficient relief because of the structural changes present, or if it is felt from the outset that palliative measures would be of doubtful value, an attempt is then made to flatten the arch by operation. Formerly I tried to do this either by means of osteoclasis, simple tenotomy of the plantar fascia with wrenching, or excision of the plantar fascia, but these methods have all been replaced by the Steindler operation. I began doing this method by means of a horse-shoe incision around the heel, as suggested in [4] Steindler's first paper. This gave an excellent exposure but there were two serious drawbacks; one, an occasional sloughing of the wound edges, and the other, a delay in the after-treatment; the sole could not be stretched for some time for fear of tearing open the newly-healed wound. Steindler himself gave up the horse-shoe incision and now operates through an incision on the inner side of the foot, extending from the back of the heel to a point in front of the anterior end of the os calcis. I find this route gives good access to the attachment of the plantar fascia, but the ultimate exposure is not as good as that through the old incision. By retracting the edges of the wound the fascia can be easily exposed from the inner side, although the removal of obtruding fat helps considerably. The knife cuts right across the fascia on to the os calcis, the tendinous band is then stripped forward by a curved rougine, taking with it its associated muscles. The long plantar ligament is then exposed. It is very adherent to the bone and may be a little difficult to lift and incise. This having been done, the foot is then wrenched and the arch flattened; the toes are manipulated, the wounds closed and dressings applied. The foot is then encased in plaster of Paris with a slotted sole-plate incorporated. The next day chamois leather tapes are applied, binding the toes; and each day afterwards the tapes are slackened to allow the toes to be stretched, and the tapes are re-applied. Stitches are removed in fourteen days, and then daily massage and stretchings of the fascia and toes take place; a slotted sole-plate is worn in the interval. The patient commences to walk in a soft shoe three weeks after operation. The intrinsic muscles of the foot are electrically treated by means of the faradic foot-bath, and the patient is also taught toe exercises.

In a little while the state of the feet is sufficiently improved to enable the patient to wear ordinary shoes, to the soles of which an outside wedge is added. He is encouraged to continue the exercises and wear the sole-plates at night for several months.

If, when the stitches are removed, the toes are still contracted, the extensor tendons are tenotomized and continuous treatment with the sole-plate is prolonged for three weeks or more before exercises begin.

(3) *Pes Cavus of Severe Degree.*

For the type of pes cavus which constitutes a gross bony deformity, and where every step is a painful one, an attack on the soft parts alone is unlikely to bring relief. In all such cases the Steindler operation should first be performed, to be followed later by a bone resection. I prefer a cuneiform *tarsectomy* at the maximum point of the convexity of the arch, which is usually in front of the astragalus (see fig. 2). A linear incision is made in the middle of the dorsum of the foot; the extensor tendons are drawn aside and a carefully planned wedge extending right across is removed. This is done without respect to the bones traversed, but I find that the cuneiform bones and scaphoid suffer most, although it is surprising what a small wedge will suffice for the purpose. It is important at the outset to aim at the removal of as little bone as possible, for a larger wedge than is necessary will not only flatten the arch,

but produce a bad calcaneus deformity as well. The following case is an example of a severe pes cavus in which this operation was performed :—

G. W. (see fig. 3), aged 23, admitted to hospital with severe double pes cavus. The state of the feet began to trouble him at the age of 6, and it has become gradually worse. Two uncles and one cousin have similar feet. Dr. Feiling diagnosed progressive muscular atrophy. On March 5, 1925, a Steindler operation was performed on the left foot, followed on April 16 by a cuneiform tarsectomy. The foot was encased in plaster until June 30, when a boot was applied. On September 17, 1925, a Steindler and wrenching operation was performed on the other foot, which, however, was not quite so badly deformed as the right one. And although not as flat as its fellow, the improvement was such that no tarsectomy was done, but exercises, massage and electrical treatment were given. He left the hospital on December 1, 1925, wearing boots with varus wedges and provided with sole-plates for night wear. He was last examined on February 22, 1927, and the correction is maintained. Before the operations he was unable to walk at all bare-footed, and had to give up work because of pain in his feet. He is now comfortable, and on his feet all day, being employed as a manual labourer.

Osteotomy.

Where the bony deformity is not very severe, and yet there is a good deal of dropping of the head of the first metatarsal, much improvement can be effected by a method shown to me by Mr. Tubby. A few weeks after the sole has been stretched by Steindler's operation, an osteotomy of the first metatarsal is performed at the junction of the anterior third and posterior two-thirds. The corrected foot is encased in plaster for six weeks and then treated with massage, exercises, etc.

Tendon Transference.

In combination with a plantar fascia operation, one has sometimes cut the extensor longus hallucis and re-attached it to the first metatarsal through a tunnel just behind the head and joined the distal end to the extensor brevis hallucis.

It must be admitted, however, that the result is often disappointing, the new attachment of the tendon exercising very little influence in counteracting the dropping of the anterior part of the foot, and the interphalangeal joint of the great toe becoming, if possible, more flexed.

I have also combined this with the attachment of the extensor of the little toe to the fifth metatarsal, but the result is not impressive. I have no experience of attaching all the long extensors to their respective metatarsals; it is probably a tedious procedure, but if effective would be worth the trouble; American surgeons (Sherman and Ryerson) speak well of it.

Arthrodesis.

In essential cavus there is little scope for arthrodesis. There is one condition, however, in which it is most effective, and that is flexion of the great toe with a painful dorsal corn. The joint is approached through a dorsal longitudinal incision, the tendon is split in its length, and each half retracted. The ends of the bones are removed preferably by an osteotome; a bone forceps is apt to crush and diminish the surfaces of contact. The terminal phalanx is put up slightly hyperextended, and a narrow metal splint, coming well on to the sole, is applied for from four to six weeks.

In the mixed deformity, such as cavo-varus, which is apt to occur in infantile paralysis, an arthrodesing operation, after correction, is valuable in securing stability.

48 Rocyn Jones—Pugh: *The Treatment of Pes Cavus*

For this condition I have often performed Hoke's triple arthrodesis with a transference of the tibialis anticus to the cuboid to counteract inversion, and the result is usually very satisfactory. Where, however, the paralysis is so severe that both the tibialis anticus and the extensor longus digitorum are affected, so that there is little or no power in dorsiflexing at all, the dropping of the anterior part of the foot presents a difficult problem, for it is apt to persist or recur in spite of a fascia-sliding operation and arthrodesis, although it may not be much in evidence when the patient stands. This type of pes cavus is to my mind the most difficult we are called upon to treat. In a review of my cases made recently the results in this type of case were found to be the least favourable.

But putting these severely-paralysed cases aside, it may be said that the after-results of treatment on the lines indicated for pes cavus is on the whole distinctly satisfactory, both from the point of view of the appearance of the foot, the improvement in walking, and the comfort of the patient.

Conclusions.

(1) A pure cavus deformity is not very common; but associated with other deformities it occurs very frequently.

(2) The ætiology is often obscure. The contributory causes may be divided into postural, paralytic and traumatic. The great majority of cases are paralytic or paretic.

(3) Symptoms usually develop during adolescence, although the exciting cause may be some remote illness.

(4) There is dropping of the foot due to weakness or paralysis, with resulting secondary contracture of the plantar fascia forcing the bony arch to expand upwards during growth.

(5) There are two clinical types: talipes arcuatus and talipes plantaris.

(6) The form of treatment depends upon the degree of cavus. In slight cases, proper boots, splinting and exercises are enough. In more severe types, Steindler's operation and wrenching are performed. This operation is the most important recent advance in the treatment of pes cavus. In very severe cases a fascia-sliding operation and a cuneiform tarsectomy are necessary.

(7) Proper after-care, in the way of splinting, exercises, electrical treatment, and footwear, is important.

References.

[1] Duncker, F., "Pes Cavus and Similar Progressive Deformities as a Result of Spina Bifida Occulta," *Zeit. für Orth. Chir.*, 1913, Bd. xxxiii, Heft 1-2. [2] Parker, C. A., "Hollow Foot—Pes Cavus," *Trans. Section on Orthop. Surg. Amer. Med. Assoc.*, 1913, p. 147. [3] Fisher, F. R., "On Paralytic Deformity of the Foot," *Lancet*, 1889, i, pp. 112, 142, 214. [4] Steindler, A., (1) "Operative Treatment of Pes Cavus," *Surg. Gyn. and Obst.*, May, 1917, p. 612; (2) "The Treatment of Pes Cavus," *Arch. of Surg.*, 1921, March, p. 325.

Dr. Gordon Pugh (President)

said that in connexion with ætiology, Mr. Rocyn Jones mentioned particularly the mild pes cavus which was apt to follow an acute illness; that, he (the President) thought, was not sufficiently appreciated. Often he found that children who had been kept in bed for a long time on account of some disease suffered from mild pes cavus. Every child who had to keep its bed longer than a few days should have the weight of the bed-clothes taken off the feet by means of a low cradle, one only

just high enough for that purpose. Also, such children should perform ankle-joint and foot exercises several times daily. Those measures were very effective in preventing definite, though mild, pes cavus in children who had been kept in bed for some time.

Mr. NAUGHTON DUNN

said that there were several types of pes cavus in which it was possible to say definitely what was the cause of the deformity. These were the cases as to which the line of treatment was generally agreed upon, and it was usually very effective.

The cause might be postural, in that the malposition had begun to occur during an illness, when the cavus deformity might or might not be accompanied by some shortening of the tendo Achillis. Another type of case in which the cause of the deformity was easily explained occurred when the dorsiflexors of the ankle, i.e., the tibialis anticus and peroneus brevis muscle, were weak. Dorsal dislocation of the toes was the result of the effort of the extensors of the toes to clear the foot from the ground in walking. This was followed by dropping of the fore-part of the foot, dorsal dislocation of the toes, with secondary contracture of the plantar structures.

Deformity in these types of pes cavus could usually be completely corrected by division of the plantar fascia and the structures attached to the os calcis, either by the tenotomy knife, or by the open operation suggested by Steindler. The important point in the operation was to bring about complete over-correction of the deformity by means of a wrench. Over-correction of the deformity was necessary so as to enable us to fix the foot in the normal position in plaster without risk of the formation of pressure sores.

If true shortening of the tendo Achillis was also present this structure might be elongated, but only sufficiently to allow the foot being brought to a right angle. If more were done in that way there was a risk of calcaneus deformity following later.

The deformity having been corrected, if there was muscle weakness in the anterior tibial group, tendon transplantation might be helpful. If the toe extensors were active, altering their insertion to the heads of the metatarsal bones would often be effective. In other cases in which there was general laxity of the joints of the foot, with practically no power in the anterior tibial group of muscles, it was sometimes better to resect the mid-tarsal joint so as to prevent recurrence of the deformity, and allow the fore-part of the foot to clear the ground in walking.

Another type of cavus deformity was that associated with calcaneus. This was usually most marked when the tendo Achillis was paralysed, while the posterior tibial and peroneal muscles retained their power. The deformity occurred because these tendons, passing behind the ankle and the fore-part of the foot, in their effort to raise the heel, merely drew the pillars of the arch together and produced a progressive cavus deformity of the sole. In these cases operation on bone was necessary to correct the deformity, and it was also wise to re-inforce the paralysed tendo Achillis by tendon transplantation. A slide was shown illustrating the operation which he practised in those cases.

The type of cavus deformity in which he found operation most difficult was that in which the onset was insidious, progressive, and associated with inversion of the os calcis. By division of the plantar fascia and the structures attached to the os calcis good correction could be secured by thorough wrenching, but there was a great tendency to relapse in these cases.

In the early cases he had found it an advantage to divide the tibialis posticus

50 Dunn—Ollerenshaw: *The Treatment of Pes Cavus*

tendon with a view to preventing recurrence of inversion of the os calcis which was often the most troublesome part of the deformity. In more established cases division of the internal lateral ligament and of the strong ligaments between the os calcis and the astragalus might be necessary. If the articular facets were so altered that a good weight-bearing position could not be maintained, arthrodesis of this joint might be necessary.

Mr. Robert Ollerenshaw.

I do not propose to refer to talipes calcaneo-cavus or other compound variety of cavus. Although we are met to consider treatment only, I nevertheless briefly discuss the ætiology of pes cavus, as a proper appreciation of the cause is so important in arriving at the correct line of treatment. I think we all agree that, with extremely

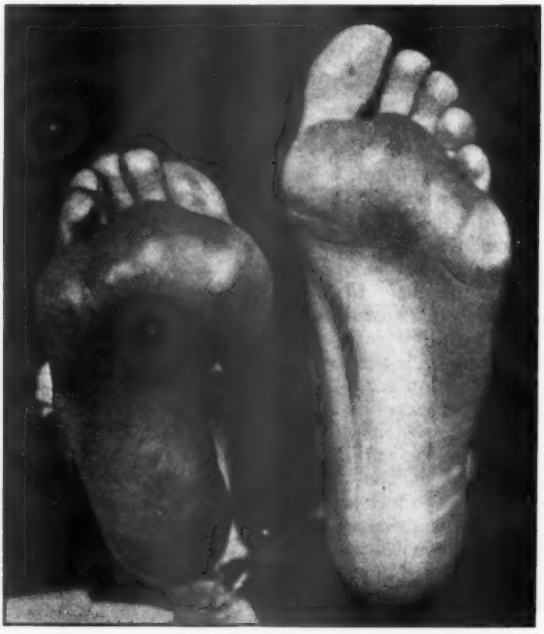


Fig. 5.

few exceptions, pes cavus is due to a disturbance of the normal muscular balance between the extensors and flexors of the ankle and toes. Whether we accept Duchenne's theory, or the suggestion that the over-pull of the extensors of the toes is an attempt to compensate for a deficiency in the normal dorsi-flexors of the ankle, matters nothing, we all observe that the cavus is produced by the over-action of the long extensors of the toes and that, as a result of this, the heads of the

metatarsals and the anterior arch become more and more depressed, and the foot more and more shortened, until a real shortening of the foot occurs. Looking at the sole of such a foot we find the appearance seen in fig. 5. This photograph was taken immediately before operation ; the darker colour of the cavoid, right foot, is due to iodine preparation having been applied. Since 1921 I have dealt with such cases entirely by transplantation of the five long extensor tendons. These are passed through a tunnel drilled from side to side through each metatarsal neck, and the tendon sewn back to itself, forming a loop. The interphalangeal joint of the hallux is always arthrodesed to prevent flexion, with its associated pressure troubles. This arthrodesis is not necessary in the other toes because in these the extensor brevis, by

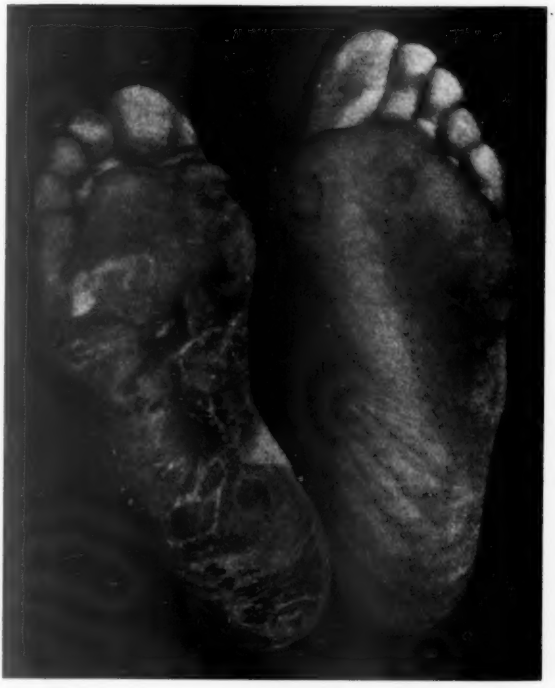


FIG. 6.

its insertion into the dorsal expansion, prevents flexion. In all cases the plantar fascia is divided, but no deeper structures in the sole are interfered with. A toe splint is fixed to the hallux and a metal shoe keeps the foot at rest at a right angle for six weeks. The effect of this transplant is excellent. The transverse arch is raised and the foot is, in consequence, lengthened. Fig. 6 shows the same foot as fig. 5, two months after operation. The appearance is very striking and the improved function is just as impressive. This is by no means an exceptional, but merely a good average result. I have performed this operation thirty-three times in the past five years. The operation has been criticized as being tedious, and I agree that it takes

FIG. 7.

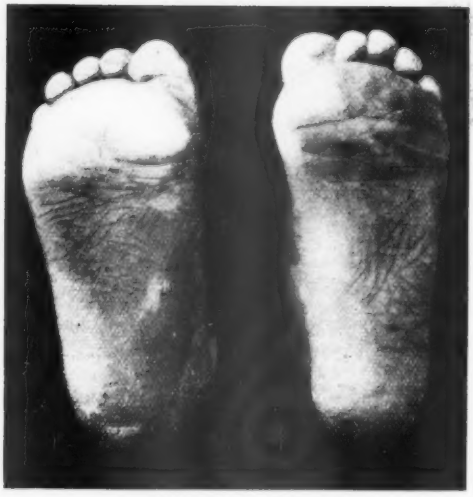


FIG. 8.

about an hour to complete. In my opinion, however, the results so far surpass those obtained by any other method, that such an objection should not be considered. (Figs. 7 and 8 show another case before operation and two months later.)

In the whole of my series of cases I have not had one patient who has not been very gratified with the result of the operation, and many of the patients are working well at regular standing occupations. The patient whose feet are shown in figs. 5 and 6 has worked for a year as a sorter at the General Post Office without the slightest trouble. I certainly consider tendon transplantation to be the operation of choice in pes cavus.

Mr. Whitchurch Howell

said that there were four points he wished to discuss.

(1) *Division of the Plantar Fascia.*—This should be done less and less, and only in the mild cases if a metatarsal bar were not sufficient. Such a case was one in which a boy played football well one season, but the following season he found he was tripping. It was then found that there was a slight contraction of the plantar fascia. In a case of this kind division of the fascia with Jones's tenotome, and manipulation with a wrench, keeping the patient in a walking plaster for six weeks, and the use of a metatarsal bar afterwards, would be sufficient.

(2) *Transference of the Extensor Longus Pollicis through the neck of the Metatarsal Bone should be condemned.* He regarded it as anatomically unsound.

(3) *The Use of a Modified Steindler's Incision.*—He had been employing this for the past five years; instead of making the original horse-shoe incision, he made two incisions, one on each side of the os calcis, so that in the case of a child of 10 to 12 years of age about three stitches were put in afterwards. One thus saved the heel-flap, and every structure it was the surgeon's wish to divide he could see; division was done with the knife, not with the raspatory. In that way one could see that one obtained a gap of half an inch, in certain cases of pes cavus. It was completed with a Thomas's wrench. No Steindler's operation was performed unless a lateral view was taken of both feet; the reason being that the difficult cases of pes cavus were those which were just short of requiring the bone operation. By a side-view skiagram of both feet one could see the relation of the longitudinal arch of the foot in comparison with the base.

(4) *A Steindler and a bony operation* should not be performed on one and the same case. Only one or the other operation should be carried out. The after-treatment consisted in the application of a walking plaster with felt across the dorsum of the toes for three weeks, the stitches being taken out on the tenth day. The patient was allowed to walk in plaster on the fourteenth day. At the end of the third week the plaster was removed, and a felt metatarsal bar was put into a stout leather-soled shoe or boot. At the end of the sixth week the patient was allowed to walk in boots made to measure, with a concealed metatarsal bar, and, if necessary, a varus wedge.

Mr. H. A. T. Fairbank

said there were only two matters to which he wished to refer. One had reference to the Steindler operation or rather a modification of it. For some years, he (the speaker) in the treatment of a foot with severe congenital or other form of cavus, had been in the habit of making an incision on the inner side of the foot in order to divide the ligaments which the Steindler operation did not enable one to reach; then of making another incision on the inner side of the heel, so as to divide the

54 Fairbank—Verrall: *The Treatment of Pes Cavus*

structures more or less after the Steindler method. He believed it was Mr. Trethowan who had communicated this idea to him. It was not advisable to divide the ligaments between the internal cneeiform and the first metatarsal, because if that were done subluxation of the base of the metatarsal into the sole might occur. He left that joint untouched. But division of the strong ligaments of the astragalo-navicular and navicular-cuneiform joints was of great use in enabling one to carry out correction in a bad case.

It was doubtful whether, in performing the Steindler operation, one should pare off the structures in the sole from the os calcis deep to or superficial to the periosteum. He had seen some serious lumps of bone in the sole after the Steindler operation. Dr. Steindler's assistant, Dr. Klein, was in this country last week, and he (the speaker) asked him what Dr. Steindler did. The answer was, a subperiosteal operation, but not at the tuberosities of the os calcis; he cut just superficial to the periosteum over the front of the tuberosities, and as soon as he had cleared these, the rest of the separation was done subperiosteally. If performed in this way, no uncomfortable mass of bone developed in the sole.

The only other question to which he wished to refer was that of doing transplantation of tendons to prevent relapse of the cavus. In a fair number of cases he had done transplantation of the extensor tendons into the heads or necks of the metatarsal bones, and had been rather pleased with the results. If all cases were carefully followed up, probably there would be forthcoming a certain number of good and a certain number of bad results, after every method of correction. He had put the whole five extensor tendons into their respective metatarsals, and had done what he thought was rather better, namely, put the extensor longus hallucis into the first metatarsal, the next two tendons into the second, and the next two into the third. He had also put the first three tendons into the first three metatarsals and simply cut the other two. He was not prepared to say as yet which of these gave the best results. There was, however, something to be said for concentrating on the second and third metatarsals. The transplantation of the extensor longus hallucis was a very useful method of dealing with the cocked big toe. In the worst cases excision of the interphalangeal joint was necessary, but in most cases if one inserted the extensor longus hallucis into the first metatarsal, and the distal cut end of this tendon into the periosteum of the proximal phalanx, the results were good. In the case of younger children he had put the extensor hallucis into the second or third metatarsals when there was some varus present, and he had been fairly pleased with the results.

Mr. P. J. VERRALL

said he had carried out the five-tendon transplantation more than a dozen times, and had been very pleased with the results.

With regard to the Steindler operation, he did it in the way many of his colleagues did, making an incision of about an inch on the inner side and, using the knife, cutting down on the bone above the tuberosities of the os calcis; he again used the knife to separate the lower part of the heel from the sole; he then employed

the periosteal elevator above the incision, so producing exactly the result Mr. Fairbank mentioned. It was really doing a subperiosteal separation above the point of the incision.

He was very disappointed with the single first metatarsal transplant. In certain cases, such as those to which Mr. Fairbank had alluded, in which there was a drop of the first metatarsal head and not much else, it was rather promising; but, on the whole, he was not pleased with it. The five-tendon transplantation, though tedious, was one of the most satisfactory of all the operations, combined with the Steindler operation at the same time.

He belonged to a school which refrained from bone operations, except in the rarest kind of cases, for any form of pes cavus. One could divide tendons and ligaments, by wrenching and over-correction, and with a Jones' wrench the sole could be made to bulge down. He very much disliked bone operations.

He was interested in hearing that Mr. Naughton Dunn did his own valuable operation in cases of pes cavus; it seemed to be a very useful proceeding where a bone operation was inevitable.

Mr. Rocyn Jones (in reply)

said he was very gratified by the discussion which had taken place. He agreed that it was important to prevent the development of pes cavus in the way indicated by the President.

He probably had not made his meaning clear with regard to the elongation of the tendo Achillis. He kept the plaster on six weeks instead of three weeks, so as to keep the part for that length of time without functioning.

With regard to bone operations, it was true that these were seldom required. In only four cases of his series had the need arisen for a bone operation. In one or those cases which he had depicted on the screen, he did not see how the condition could have been overcome except by a drastic operation, either a bone operation of one like Mr. Dunn's.

Mr. Dunn had mentioned inversion of the heel. In severe cases that was a bugbear. He had excised the internal lateral ligament and wrenched the foot, everting the heel.

Mr. Whitchurch Howell had mentioned that it was unnecessary to do a bone operation. But there were some cases in which that was imperative; the amount of bone removed, however, was very small. He thought it was better to start off with the Steindler operation for the reason that even after the bone operation had been done the foot could not be flattened, owing to the violent pull on the pillars of the arch by the powerful plantar fascia.

He was very glad to hear Mr. Ollerenshaw mention his tendon transference operation. It was a tedious procedure, and that was probably the reason why it had not been more extensively adopted. American surgeons and some surgeons in this country seemed to have a high opinion of it, because it prevented the dropping of the anterior part of the foot, which was so important.

With regard to Mr. Fairbank's remarks, he (the speaker) had recently had the opportunity of examining a cavus foot which had been amputated because of long-

standing poliomyelitis; the patient had perforating ulcers with extensive dermatitis, and it had been necessary to perform a Syme's amputation. The layers of small muscles in the sole were adherent to each other and it was difficult to separate the musculus accessorius from the long plantar ligament.

In the Steindler operation, after incising the plantar fascia the rougine swept the muscles forward *en masse*. With adults it was very difficult to cut the periosteum and push it forward, as Steindler suggested in his paper published in 1917. It could be done in the case of children, but not for adults without risk of tearing up bone as well. It was that which caused the post-operative exostoses observed by some American writers. He agreed with Mr. Verrall that the operation could be done without attempting to push the periosteum forward.

Section of Otology.

President—Dr. Dan McKenzie.

The Treatment of Traumatic Facial Paralysis.

By E. Farquhar Buzzard, M.D.

The principles governing the treatment of all lesions affecting the lower motor neurones must of course apply to lesions of the facial nerve, but the path of that nerve through the substance of the temporal bone, where trauma may be inflicted, creates certain difficulties which deserve special consideration. One of these arises from the obscurity which often surrounds the nature and extent of the lesion, the impossibility of determining whether in certain cases the nerve has been completely divided—a rare accident—or whether it has been lacerated, bruised, crushed or merely exposed to the injurious effects of inflammation in adjacent tissues. A second difficulty only affects those instances of complete or nearly complete division which, in the case of other nerves, might call for local surgical intervention in the form of resection and reunion operations which, for anatomical reasons, are precluded in the case of the facial nerve.

Speaking generally, therefore, a facial palsy resulting from a lesion of the nerve in its course through the temporal bone does not call for operative interference, and we have only to consider whether other measures and, if so, what measures are calculated to expedite recovery of function.

These cases of facial palsy may be roughly divided into two main groups. In the first may be included all cases which, after a lapse of three weeks from the onset of palsy, display even the slightest return of function in the facial muscles and in which the muscles do not show the reaction of degeneration to electrical tests. Returning voluntary movement and the absence of reaction to degeneration afford sufficient proof that the continuity of the nerve has not been disturbed, that function has been only temporarily interfered with, and that recovery is merely a matter of a few weeks at most, often much less. It is doubtful whether any treatment will expedite recovery in this group of cases, but gentle rubbing of the facial muscles, followed by the patient's own attempts to carry out facial movements, can be trusted to prevent undue delay. Perfect recovery, uncomplicated by contractures or spasmodic contractions, may be justifiably expected.

In this group of cases, in which the nerve does not go through the processes of degeneration and regeneration, three weeks has been given as the period of time after the onset of paralysis after which signs of returning function may be observed. This is a somewhat arbitrary estimation, as signs of recovery may be further delayed, in the absence of the reaction of degeneration, in cases in which active inflammatory disease persists at the site of the trauma. In such cases, of course, the return of voluntary facial movements may be proportionately postponed.

The second group of cases comprises those in which the reaction of degeneration is present at the end of three weeks from the date of onset. In the majority of such cases only time will show whether regeneration is possible; no evidence of regeneration will be forthcoming until after three or four months, and the question of treatment in the interval has to be decided.

36 Buzzard: *Treatment of Traumatic Facial Paralysis*

We are unacquainted with any means by which the regeneration of the nerve may be hastened. We must be content to employ treatment measures calculated to preserve the nutrition and contractility of the degenerated and atrophied muscles and to prevent the development of disfiguring contractures.

The principle of keeping flaccid paralysed muscles unstretched is not so easy to put into practice in relation to the face as it is in the case of the limbs, and the only device for that purpose is a simple apparatus designed to prevent the mouth from being drawn to the opposite side with the consequent stretching of the muscles attached to its angle and upper border. Fortunately the patient, from æsthetic motives, is inclined to inhibit facial movement, and so unconsciously co-operates with the surgeon in avoiding the risk of permanent disfigurement. The nutrition and contractility of the paralysed muscles may be preserved by massage and by compelling contraction by means of the make-and-break of the galvanic current. Probably the regular employment of such measures insures the muscles being in a fit condition to take up their activities again when their innervation has been restored and their regeneration effected. As soon as any voluntary movement is possible re-educational exercises of the muscles before a mirror is the best method of regaining normal control over the features; massage and electrical treatment are no longer necessary.

At this stage post-paralytic contracture often develops, characterized by some permanent shortening of certain muscles and by the patient's inability to discriminate in his facial expressions. Thus, in showing his teeth, the withdrawal of his lips will be accompanied by a strong contraction of the orbiculares palpebrarum, and in closing his eyes he cannot avoid raising the corner of his mouth; a smile is inevitably associated with a compromising wink.

It is doubtful whether this complication can be avoided by any means, and treatment by massage and electricity is of no value once the condition has developed. It is difficult to say for how long it is desirable to use massage and galvanism in the hope of obtaining signs of nerve regeneration; the decision must be modified by special circumstances, but, generally speaking, if there is no return of voluntary movement or of response to the faradic current at the end of six months it is useless to persevere with those methods of treatment.

We have seen this morning the results obtainable from anastomosis in cases of complete and permanent division of the facial nerve. They must be judged on their merits. Personally, my experience of such operations is very meagre and does not justify any decided expression of opinion.

Discussion.—Dr. DAN MCKENZIE (President) said that the subject of facial paralysis was near to the heart of every otologist. When the aural surgeon saw a case of facial paralysis after an operation on the ear, the first question he asked himself was, what treatment was required? Did, e.g., the question of making an anastomosis arise? He (the speaker) thought that this question could be settled, most frequently, by considering the cause of the paralysis. What kind of accident to the nerve during the operation had resulted in the paralysis? Those who saw these cases were inclined to conclude that if the nerve had been struck by a blunt instrument or curette the patient would probably recover from the paralysis, and in such cases electrical treatment aided the nutrition of the facial muscles and their consequent return to power. If, however, the nerve had been cut through by a mallet or a sharp chisel or gouge, the question of recovery was more difficult. He (the speaker) thought that most of such cases would recover if left alone. In one case Sydenham had joined the two ends of the severed nerve together; such reunion was a possibility not to be lost sight of. But when a portion of the facial nerve trunk in the temporal bone had been excised, no natural recovery would take place, and in such cases question of the anastomosis operation aroused much interest. Otologists would like to be assured that anastomosis was going to be successful; the operation was still in the experimental stage, and every effort

must be made to perfect it. At present the results were not always thoroughly satisfactory. He (Dr. McKenzie) did not think that the emotional movements had followed anastomosis, even when the operation had been most successful; both sides of the face did not duly respond to the emotions of the mind. That result, however, might occur in time. But even if only the flaccidity of the paralysed side of the face could be prevented, a great deal would have been achieved.

Mr. A. J. M. WRIGHT said he thought that the six months' limit for recovery given by Dr. Buzzard was too short a time.

Mr. SYDNEY SCOTT said he had seen cases in which the facial paralysis did not begin to disappear, in spite of electric treatment, until three years after the onset of the paralysis.

Sir WILLIAM MILLIGAN said British otologists were to be commended for the rarity of cases of facial paralysis following operation, in comparison with the number of such cases in other countries, and, from what one saw at British hospitals, it was clear that this comparative infrequency was due to the meticulous care which aural surgeons exercised in operating. When the injury did occur, it was, of course, most disfiguring.

In this connexion junior surgeons should be warned that although there was, of course, a danger of the assistant's injuring the nerve in the course of the operation, sometimes the injury arose during the after-treatment, if packing was made too tightly, or if caustics were injudiciously used. Packing the mastoid cavity should be done lightly and gently.

It was difficult to decide how long one should wait before operating, but he, too, thought the period of six months far too short. He had known the nerve to recover after a period of two years. It was essential, when facial paralysis developed, that immediate care should be taken of the facial muscles; the surgeon should anticipate nerve recovery and keep up the tone of the muscles by electrical treatment and massage.

Recently he (Sir William) had had a case in conjunction with Sir Charles Ballance, that of a child suffering from extensive tuberculosis of the middle ear, mastoid and labyrinth: the patient for seven years had been under his (the speaker's) treatment. It had not been possible to get the cavity into a condition sufficiently healthy to warrant an anastomosis operation. During the whole time the patient had had daily massage, and the muscles had been stimulated daily; finally the cavity had healed, the anastomosis operation had been performed, and function was now returning.

Opinions differed as to the type of anastomosis desirable. Whatever nerve might be selected, there was sure to be some discomfort for a time. The point to emphasize was that after anastomosis the same care should be taken as before to maintain the tone of the muscles; definite improvement might not occur for a long time. He (Sir William) had never known complete return of function after an anastomosis operation. He had, however, seen great improvement, and the patients had appeared to be satisfied.

Mr. E. WATSON-WILLIAMS said that a point not dealt with in the paper was the prognostic significance of the time at which the paralysis came on. A paralysis which came on during or immediately after an operation was likely to be much more serious than one which appeared only after two or three days. He had especially noticed that difference after labyrinthectomy, an operation about which he had felt somewhat diffident, because among numerous cases he had not had one without evidence of facial nerve damage; fortunately all the patients had recovered or were on the way to recovery.

Much gratitude was due to Sir Charles Ballance for his new as for his former experimental work on facial nerve anastomosis. Reluctance had sometimes been felt in proposing to use a motor nerve, as the results were not invariably satisfactory from the facial point of view, and might be even less satisfactory with regard to muscles supplied by the eleventh and twelfth nerves. It was helpful to know that anastomosis could be carried out with a nerve which could be spared without inconvenience by the patient.

Sir William Milligan had said that some disturbance always followed the operation. He (the speaker) had twice divided the glosso-pharyngeal nerve and on neither occasion did the patient suffer any appreciable inconvenience, though the first patient had complained for a few days that there was a little stiffness of the throat on attempting to swallow. Therefore the operation might be undertaken with the assurance that at worst it would do no harm. An astonishing discovery was that the glosso-pharyngeal, excluding the nerve to the stylo-

pharyngeus, would act as a motor nerve, whereas according to the text-books it was purely sensory. The very fine nerves to the pharyngeal plexus were not likely to survive intact, and only the sensory portion of the nerve would remain when it was anastomosed. Physiologists would have to re-write part of their story of the functions of nerves if it was demonstrable that a sensory nerve could be trained to move muscles.

Sir JAMES PURVES-STEWART said that Members were probably aware that work in connexion with hypoglossal-facial and spinal-accessory-facial-anastomosis had been done by Sir Charles Ballance, himself (the speaker) and others, twenty years ago. A clear conception of what was meant by the term "traumatic facial paralysis" was necessary. Did it include trauma during the course of an operation by the surgeon, by his assistant, or by nature, on the middle ear?

It was useless to anastomose the proximal end of a sensory nerve, such as the glosso-pharyngeal, below the level of its motor pharyngeal branches, to the distal end of the facial nerve. Further, the glosso-pharyngeal was a very small nerve, the hypoglossal was larger, and the spinal accessory was largest and easiest to find.

The nerves which gave the best clinical results when used for anastomosis were the hypoglossal and the spinal accessory, and of these two the hypoglossal was the better, because when one divided part of the spinal accessory and attached its proximal end to the distal end of the damaged facial nerve, each time the patient tried to elevate the scapula the face, owing to the action of the spinal accessory, contracted on the paralysed side, and unilateral momentary partial closure of the eye—say by a lady acquaintance in the street—was apt to lead to social misunderstandings. If, however, one chose the hypoglossal nerve for the operation, the patient had a more accurate control over half of the tongue, and male patients, at any rate, could keep the tongue at rest inside the mouth; whilst even female patients would restrain its activity when they found that their faces moved every time they tried to innervate the affected side of the tongue. The chief disadvantage of the use of the hypoglossal was the resulting hemiatrophy of the tongue, but this was overcome after a few weeks, when the patient became habituated to the new conditions.

Mr. G. J. JENKINS referred to a case illustrating an important point, namely, that an anastomosis of the seventh cranial nerve with the descendens hypoglossi might give a very satisfactory result, even when performed four years after the commencement of the paralysis. Again, cases had been known to recover function without operation three years after the onset of the paralysis. He also would be glad to know how long one should wait before operating.

Sir JAMES DUNDAS-GRANT said that at a recent meeting he had read notes of several cases of this kind. In one the paralysis had seemed to be complete, but a change for the better had taken place after the employment of digital massage. In another, the use of the anodal pole of a mild galvanic battery had had an excellent effect. This pole had been found better than the negative pole, and less likely to cause contracture. There had been a case in which traumatism was uncertain, but in which also the paralysis had seemed to be complete. He (Sir James) had used faradism merely with the object of demonstrating its uselessness, and, to his surprise, it at once restored the most active movements. In fact, the nerve had recovered but the cortical centre had "hung fire." Judging from his own experience, it seemed that it was in simple operation for acute cases rather than in the radical operation that there was most liability of damage to the facial nerve. In one case he was sure that if he had been allowed to operate earlier the temporary paralysis would not have taken place. During the radical mastoid operation the landmarks were more clearly under the surgeon's view. He (Sir James) had shown a boy suffering from facial paralysis and tuberculous disease of the middle ear and mastoid. He had been under observation several years, and though no improvement with regard to facial paralysis had been expected it had now entirely disappeared.

Mr. M. VLASTO said that facial nerve anastomosis was not without operative risk, apart from that of infection. After the complete division of the facial nerve, one step in the operation, the face on the affected side lost what little tone it had possessed, and the immediate effect was to render the paresis more obvious. Another risk had come under his notice recently, namely, paralysis and atrophy of the tongue on the side of the anastomosis supervening several days after the operation.

The cases of facial paralysis which he (the speaker) had seen were due either to pathological processes over which the surgeon had very little control, or to an error in surgical technique. He was not familiar with the type of case in which paralysis had ensued as a result of injudicious cauterization. Cauterization in his cases was carried out only at a much later stage, when the facial nerve was well protected by granulations.

He believed that the scarcity of cases of facial paralysis seen in this country might be due less to the caution of British surgeons than to their over-timidity in bevelling down the posterior meatal wall, thus increasing the difficulty of the after-treatment.

Mr. LIONEL COLLEDGE said that in the cases which recovered signs of recovery had almost always appeared by the end of four months. If no signs had appeared at the end of six months, recovery was unlikely; if none were evident at the end of a year recovery was extremely improbable. Cases which recovered spontaneously after a longer interval must be so exceptional that it was not justifiable to take them into account in arriving at a decision about the anastomosis operation. Still, the two cases he (the speaker) was now exhibiting showed that recovery could take place two or even four years after the onset of the paralysis. Sir William Milligan had insisted on the importance of treatment in the meantime, but neither of these patients had had any such treatment, so that apparently this was not essential to the recovery of the muscles.

It should be strongly emphasized that, whatever nerve was employed in the operation, the whole trunk should be employed. If a mere end-to-side anastomosis was made there could be no hope of dissociated movement.

Dr. C. P. SYMONDS said that one explanation of some of these cases of long-delayed recovery was that we were dealing with a functional perpetuation of what had been an organic disability. As Dr. Buzzard had pointed out, a patient with facial palsy learned to inhibit the movement on the other side of the face to some extent, and was, as it were, "going slowly" with the whole of his face. If one allowed the patient to be merely passive and simply treated him by massage and electricity, there was the danger that when regeneration occurred he might not exploit it adequately. Thus there was much to support Dr. Buzzard's argument that attempts at active movement should be combined with massage, the patient being encouraged frequently to practise movements in front of the mirror.

ILLUSTRATIVE CASES.

Post-operative Facial Paralysis.

By DAN MCKENZIE, M.D.

A BOY, aged 3. Schwartze's operation eighteen months ago; complete facial paralysis left, noticed day after operation.

Recovery is progressing. Note an unusual feature, the replacement of the normal closure of the left eyelid in spontaneous winking by a twitch at the angle of the mouth.

Glosso-pharyngeal Facial Anastomosis for Facial Palsy following successful Trans-labyrinthine Drainage for Meningitis.

By E. WATSON-WILLIAMS, M.C., F.R.C.S.Ed.

MRS. L. J., aged 56. Admitted February 11, 1926, with purulent meningitis; labyrinth "dead." At operation the whole labyrinth found destroyed by large cholesteatoma, through which facial nerve ran. Posterior fossa drained through internal auditory meatus; facial nerve divided. Complete facial paralysis with reaction to degeneration.

October, 1926.—Proximal end of glosso-pharyngeal nerve (excluding nerve to stylo-pharyngeus) united end-to-end to facial nerve, divided at the stylo-mastoid foramen.

40 Watson-Williams—Colledge—Gill-Carey—Layton

December 1.—Flabbiness of the cheek felt to be less, and epiphora has nearly disappeared, though no visible change. Now very definite improvement in tone of face, with some capacity for voluntary movement at the corner of mouth and in forehead. In repose asymmetry of face hardly noticeable; no change in electrical reactions.

Hypoglossal-facial Anastomosis for Facial Palsy following Mastoid Operations.

By Lionel Colledge, F.R.C.S.

Captain B., aged 32. Mastoid operation in India in 1921, followed by facial palsy fourteen days later. Further mastoid operation in India, and subsequently the wound was re-opened and grafted in London two years after first operation. No recovery of facial muscles, except for slight voluntary movement of the orbicularis palpebrarum.

June, 1923.—Hypoglossal-facial anastomosis with secondary descendens hypoglossal anastomosis. There is electrical recovery to faradism of all facial muscles.

Descendens Noni Facial Anastomosis for Bell's Palsy.

By Lionel Colledge, F.R.C.S.

Miss A. H., aged 29. Referred by Dr. A. Feiling on account of Bell's palsy, showing no improvement after four years. Reaction of degeneration in all facial muscles of left side. Descendens noni facial anastomosis; descendens found to be appreciably smaller than facial. Peripheral end of descendens implanted into the side of hypoglossal. A year later electrical recovery of all facial muscles and muscles supplied by descendens. No paralysis of tongue.

Spinal Accessory Facial Anastomosis for Facial Palsy during Acute Destruction of Labyrinth.

By C. Gill-Carey, F.R.C.S.Ed.

Mrs. A., aged 29. Acute mastoid, 1924. Nerve paralysed before operation, no recovery. Radical operation, January 1925, did not heal. Labyrinth fell out as a sequestrum, immediate healing. Spinal accessory divided below nerve to sterno-mastoid and joined end-to-end to facial (Mr. C. H. Fagge), October, 1925.

Facial Palsy Five Years After Injury.

By T. B. Layton, D.S.O., M.S.

James A., now aged 16. April, 1922, complete facial palsy following attempt at radical mastoid. Injury to nerve was outside skull. Radical mastoid performed later. Some recovery of power.

Section of Otology 41

Some Points in the Diagnosis and Localization of Cerebral Abscess.

By C. P. SYMONDS, M.D.

IN this paper I shall confine myself to cerebral abscess arising from suppuration in the ear, and situated entirely within the dural membrane. I shall only refer briefly to those symptoms and signs of abscess in the cerebellar and temporal lobes which are of chief value in making an early diagnosis and localization. I shall then describe in greater detail two less common conditions, the superficial abscess, and the localized non-suppurative encephalitis.

CEREBRAL ABSCESS.

Temporal and Cerebellar.

In the early stages the symptoms are, as a rule, those common to retained pus elsewhere; fever, a rapid pulse, general malaise and anorexia. Headache may be evident but is not commonly severe. This phase may lead directly to an unrestricted suppurative encephalitis with the rapid development of severe headache, vomiting and drowsiness, as the result of increased intracranial pressure.

More commonly the suppurative process becomes localized as an abscess cavity with tough walls, through which there is little septic absorption. There is, therefore, no diffuse encephalitis, and the abscess produces signs and symptoms which depend upon its situation rather than its pathology.

In this, the common variety of cerebral abscess, the signs of increased intracranial pressure are, in the early stages, limited to occasional headache, by no means always severe, but often experienced on waking, and increased by stooping and coughing. Severe headache, vomiting, drowsiness, and slowing of the pulse, when they develop in such a case, probably mark the occurrence of mechanical obstruction to the outflow from the ventricles with internal hydrocephalus, and may be terminal in their onset. I would emphasize this point in view of the teaching in some quarters still current that headache and drowsiness, vomiting and slow pulse are the cardinal signs of cerebral abscess. In the variety of abscess that I am now discussing this is no more true than it is of cerebral tumour.

A recent case of cerebellar abscess will serve to illustrate this point. A man of 21, who had had chronic otitis media for many years, was admitted to hospital on account of occipital headache and profuse discharge from the left ear. He had been at work up till admission. Operation was performed the same day and pus evacuated from the mastoid. On the following day he made no complaint of headache, was alert and sensible, but was observed by the surgeon to have nystagmus on looking to the left and some inco-ordination of the left arm. The next day he complained suddenly of severe headache, began to vomit, and became drowsy. When I saw him twenty-four hours later his pulse had slowed from 75 to 55. He was incapable of speech and almost unconscious, being with difficulty roused to hold his arms out. When he did so the left fell rapidly away to the bed. There was bilateral ankle clonus and both plantar responses were extensor.

At the operation, two hours later, an ounce of pus was evacuated from the left lobe of the cerebellum. Within thirty-six hours the signs and symptoms of increased intracranial pressure had disappeared. The plantar responses were flexor, there was no complaint of headache, and he was as alert and bright as he had been on admission. The size of the abscess suggested that it had been present for two or three weeks.

In the early stages, therefore, we have to rely chiefly for diagnosis upon the signs of local damage caused by the abscess, together with complaint of intermittent headache. If the symptoms have supervened upon a mastoid operation and a full history is available, an account of fever, malaise and rapid pulse-rate persisting for a

week or so after the operation, is also of value. Of the additional information to be obtained from lumbar puncture I shall speak later. The localizing signs of cerebral abscess may be considered under three headings: (1) cerebellar, (2) left temporal, (3) right temporal.

(1) Of the signs of *cerebellar* abscess in the early stages suboccipital headache is of some importance, but in the later stages, when internal hydrocephalus is developing, the headache is commonly bifrontal or general.

The abscess is usually situated deeply within the lateral lobe. Corresponding with this situation the symptoms are those of inco-ordination in the movements of the limbs on the same side, which is best shown in the upper limb. Disturbance of equilibrium and reeling gait are less common or later developments.

The simplest method of examination is by the finger—nose—finger test. On the affected side there is deviation from the line of movement, and a tendency to under- or over-shoot the mark, giving a general impression of clumsiness. Of the other tests, one of the best is falling away of the affected limb when support is withdrawn by the observer from beneath the outstretched hands of the patient.

The other sign of value is nystagmus. This is sometimes said to be absent by the otologist when its presence is recognized by the neurologist. The tendency of the eyes is to swing away from the side of the cerebellar lesion. This will show best when the patient is asked to look away from the direction of the spontaneous deviation, that is, towards the side of the lesion. If he is then asked to fix his gaze on the observer's finger, the eyes gradually—it may be quite slowly—swing back towards the mid-line. This spontaneous movement the patient may or may not correct. If he does so the quick component is added and nystagmus results. If there is no attempt at voluntary correction, one cannot say that nystagmus is present, but one may speak of an unwillingness in conjugate deviation towards one side, which as a unilateral sign is of almost as great localizing value as the complete nystagmus.

(2) In abscess of the *left temporal* lobe in a right-handed person the localizing sign of outstanding value is aphasia. If the abscess be situated far forward in the lobe it may not be present, but in the commoner site in the middle or posterior thirds it is an early sign, and easily elicited if the right method is employed. It is perhaps unnecessary at a meeting of this Section to emphasize the fact that a patient may be suffering from a definite aphasia and yet be able to converse without apparent difficulty and take part in the ordinary social life of a ward full of patients without the aphasia being discovered until the appropriate method of investigation is employed.

For instance [1], a boy aged 16, who had been under observation in hospital for a week, was suspected of having a left-sided cerebral abscess. The house surgeon, who was a good observer, had written a full report in which he stated that there was no aphasia. He had rightly concluded on other grounds that an abscess was present. We made a collection of a dozen heterogeneous objects and asked the patient to name them. Books, matches, pen, watch and half-a-dozen others were named correctly. There were only two failures. When shown a key he could describe its use but could not name it. He rejected all substitutes and promptly accepted the word "key" as correct; so also with a plate. He said it was to eat off, and made of china, but could not name it. As before, he rejected substitutes and accepted the correct name when it was given to him. The abscess was found in the middle of the left temporal lobe and he made a complete recovery.

The other signs of *temporal* lobe abscess are common to both sides. They are the only signs of a temporal abscess on the right side in a right-handed person. Of these there are three which are of practical value. The first is a slight weakness of the opposite side of the face, mostly of the lower half, which may be present when the patient talks or smiles, but is less apparent when he responds to the usual test of showing the teeth at command.

The second group of signs are those which indicate pressure upon the pyramidal tract—absent or diminished abdominal reflexes, increased tendon-jerks or an extensor or doubtful plantar response.

The third sign I have placed last because it is less constantly present. When found it is of the greatest value—I refer to the defect in the visual field caused by involvement of the optic radiation. We owe to the anatomical researches of Adolf Meyer the knowledge that the optic radiation, as it leaves the thalamus, at first sweeps downwards and forwards and extends well forward into the temporal lobe before it turns back beneath and around the lateral ventricle to reach the occipital cortex. In this situation its strands are divided into two bundles. inferior and superior, which correspond with the upper and lower quadrants respectively of the visual field. The otitic abscess, as a rule, lies in the lower part of the temporal lobe, and is therefore likely first to affect the inferior bundle of the radiation, producing a homonymous defect in the superior quadrants of the opposite fields. This may be the only definite sign of a right temporal abscess, and the importance of examining the visual fields when such an abscess is suspected cannot be overrated. The examination can be made at the bedside in a few minutes with sufficient accuracy to discover a gross defect, even in a child who is seriously ill, as the following instance sufficiently illustrates:—

A girl aged 7 was admitted to hospital with a six weeks' history of intermittent headache, culminating in vomiting and drowsiness. There was a family history of phthisis, and a brother and a sister of the patient had died of tuberculous meningitis. There was a history of old-standing bilateral chronic otitis media. On examination the child was drowsy and occasionally put her hands to her head as if in pain.

Definite cervical rigidity was present; no skin or tendon reflexes were obtained.

Lumbar puncture gave a clear fluid which contained 32 cells per c.mm., all lymphocytes.

The provisional diagnosis was tuberculous meningitis. Rough examination of the visual fields, however, revealed a complete left homonymous hemianopia. Operation was performed a few hours later, and a large abscess discovered in the right temporal lobe, the pus containing streptococci. The child subsequently died of meningitis.

The Cerebro-spinal Fluid in Cerebral Abscess.

The fluid in a case of cerebral abscess is clear and contains a slight excess of cells, varying in my series of cases from 18 to 95 per c.mm. These are mainly lymphocytes. The protein content is increased—the chloride content is normal and sugar-reducing bodies are present. So long as the abscess remains localized this is the usual picture.

A preponderance of polymorphs in the fluid, with diminution of chlorides and absence of sugar reduction is usually associated with clinical evidence of meningitis and means that the abscess is leaking either into the ventricular system or into the subarachnoid space. The change in the cerebro-spinal fluid in such a case may be rapid.

For instance, in a patient with a right temporal abscess, who was mentally alert and whose general condition was fair, the fluid was clear and contained 32 lymphocytes per c.mm., ·07 per cent. protein, and ·71 per cent. chlorides. Next morning she suddenly became collapsed and unconscious. Lumbar puncture was done at once and gave an opalescent fluid full of polymorphs, which were not, however, counted. This patient was operated upon, but died three weeks later of meningitis. At autopsy both ventricles contained pus, and the cavity of the right ventricle was lined with granulation tissue. The moment of collapse probably marked the occurrence of leakage into the ventricle.

Estimation of the pressure in the cerebro-spinal fluid may also be of value in diagnosis, for a definite rise of pressure may sometimes be found preceding complaint of severe headache or other signs of increased intracranial pressure.

44 Symonds: *Diagnosis and Localization of Cerebral Abscess*

Superficial Abscess.

Three years ago I showed before this Section, together with my colleague, Mr. W. H. Ogilvie, a case [2] in which, following otitis media and mastoiditis, a localized collection of pus formed upon the surface of the brain beneath the arachnoid membrane in the neighbourhood of the Sylvian fissure. This was successfully drained and the patient has since remained well. I have recently met with a similar case.

A child, aged 5, was admitted to hospital with one week's history of left otorrhœa; a mastoid operation was done the same day. At the operation the dura in relation to the roof of the antrum was injured. For ten days after the operation the child seemed well and was allowed up. On the eleventh day after operation, at 4 a.m., she had an attack of right-sided Jacksonian epilepsy. Thereafter, at short intervals, she had many more. I saw her at 11 a.m., when she was in a constant state of right-sided clonic convulsions, so that further examination was impossible. Lumbar puncture had been done at 8.30 a.m. The fluid was clear and contained a slight excess of lymphocytes.

The argument (1) from the epileptic attacks was that she had a cortical and, therefore, a superficial lesion; (2) from the spinal fluid that the infective focus was shut off from the general subarachnoid space. Mr. Gill Carey, who operated the same afternoon, made the following note:—

> "There was a small patch of granulations present on the dura in relation to the roof of the antrum (the place where the dura was accidentally injured, but not penetrated, at the original operation). Watching these granulations carefully I saw a bead of pus come out, and on enlarging the opening with sinus forceps a quantity of pus shot out under considerable pressure. The amount of pus was enough to fill the cavity of the mastoid, roughly a drachm. There was no question of opening the dura, as it was already open. I should say, however, that the collection of pus was very superficial, as the cavity was a very shallow one. At the dressing after operation the forceps met with resistance after entering about ¼ in."

After operation there was no recurrence of the fits. The child remained unconscious for two days—presumably an exhaustion effect of the epilepsy. There were no signs of generalized meningitis. When she was fit to be examined she had a right hemiparesis with aphasia and apraxia, which gradually cleared up. The wound healed in five weeks. Six months after this she appeared in good health, but was left with slight weakness of the right arm and leg.

I take it that in a case of this kind the pathway of the infection from ear to brain has for some reason become blocked at a point at which it has already become shut off from the general subarachnoid space, and would more usually progress into the cerebral substance. The infective process then spreads laterally in the plane in which it has become arrested. The process is slow, and in the neurological sense "silent," until it reaches above the Sylvian fissure to the face-arm area of the motor cortex.

Such cases are probably rare—their immediate recognition is of obvious importance. The danger is lest the convulsions be interpreted as the first sign of a generalized meningitis, and the chance of immediate operative relief be missed.

The distinguishing features are the sudden onset of convulsions or paralysis of cortical type and the findings in the cerebro-spinal fluid.

Localized Non-Suppurative Encephalitis.

In the course of formation of every cerebral abscess there must be a pre-suppurative stage of inflammation in which there are engorgement, exudation and swelling without necrosis. I would suggest that, as in other tissues, the inflammatory process may become arrested at this stage and be resolved without pus formation.

That such a localized non-suppurative encephalitis may occur in relation to otitis media is perhaps doubtful. I put forward the hypothesis for discussion. It seems

to me the most probable explanation of cases in which the signs of a cerebral abscess are at one time present but disappear without the evacuation of pus. I have had three such within my own experience.

The first was that of a boy who had a right-sided otorrhœa and mastoiditis, for which an operation was performed. I was asked to see him two or three weeks later on account of headache and drowsiness. I found slight swelling of both optic discs, which was confirmed by an ophthalmologist, and some alteration of the reflexes on the left side of the body, suggesting a right temporal lobe lesion. An exploratory operation was performed but no abscess found. As the symptoms persisted a second operation was done ten days later. No abscess was found, but the boy subsequently made a complete recovery.

The second case was that of a man with left-sided otitis admitted to hospital for headache and drowsiness. He was somewhat confused, was definitely aphasic and had a right homonymous hemianopia. He refused operation and was subsequently discharged from hospital, having completely lost his aphasia and hemianopia.

Of the third and most recent case I have more detailed notes.

A boy, aged 10, was admitted to hospital on September 11 on account of pain in and behind the right ear following an acute otitis media of one week's duration. The temperature next day was 100° F. and there was persistent pain with much discharge from the ear. At operation, the same day, pus was found in the mastoid cells and a small collection compressing the lateral sinus which was, however, not thrombosed. The dura in the middle fossa was exposed but not incised. The wound was drained. Following the operation there was a swinging temperature up in the evening between 99° and 105° F.; the pulse ranged from 90 to 120. Mentally he was noted by the sister in the ward to be odd, apathetic, not speaking unless spoken to, taking no interest in his toys. On the nineteenth day after the operation a doubtful extensor response was obtained from the left foot.

About this time he began to complain of right frontal headache, chiefly on waking in the morning. This was occasionally associated with nausea. I first saw him on October 7, twenty-five days after the operation, and found a definite extensor plantar response on the left with diminished abdominal reflexes on the same side. I made the diagnosis of right temporal abscess and operation was performed the same day. The dura was opened and the brain explored without any abscess being discovered. Lumbar puncture at the time of operation gave a clear fluid apparently under increased pressure, containing no increase of cells or protein.

Following this operation his condition remained much the same. The temperature and pulse-rate were still high, there was a good deal of complaint of headache, but, apart from this, apathy was the most noticeable feature. Six days after this second operation I found the physical signs unchanged, and in the light of my previous experiences I suggested that we might be dealing with a non-suppurative encephalitis. It was decided, therefore, that no further operation should be undertaken for the time being, but that he should be given full doses of hexamine.

About October 16, that is, five weeks from the date of admission, he began to improve, temperature and pulse fell and headache was less; he took a natural interest in his surroundings. On October 29 he seemed normal and I could no longer obtain an extensor response from the left foot. He has since remained well. It is, of course, possible that an abscess may yet be present, but I am inclined to the opinion that my provisional diagnosis of non-suppurative encephalitis will prove correct.

The point to which I want to draw attention in these three cases is that the encephalitis was in each sufficiently well localized to cause physical signs resembling those of a cerebral abscess. All were diagnosed as such, and the only one of the three who escaped operation was he who refused it. I have been interested to find, in a recent report [3] from the Mayo Clinic, by A. W. Adson, an account of three similar cases. All three were in children whose symptoms developed after otitis media. The first had signs of a right temporal abscess, with papillœdema, and progressive left facial weakness. The second also had papillœdema, with aphasia, right homonymous hemianopia, and right-sided weakness—the classical signs of a left temporal lesion. Both were explored, the first four, the second nine, weeks after the

46 Symonds: *Diagnosis and Localization of Cerebral Abscess*

commencement of headache had first suggested the presence of intracranial trouble. In neither case was any abscess found. Both ultimately recovered. The third case, also, with signs suggesting a left temporal abscess, was watched for some weeks and the patient recovered without operation.

Adson offers no means of clinical distinction between what he calls the pseudo-brain abscess and the true. But the point of importance to be gained from these observations is that when the general and localizing signs of cerebral abscess are present a negative exploration does not *necessarily* mean an abscess missed. In such a case, therefore, it may be wise to await the possibility of spontaneous cure before proceeding to a secondary exploratory operation.

REFERENCES.

[1] RICHARDS, E. H., "A Case of Temporo-sphenoidal Abscess secondary to Otitis Media," *Guy's Hospital Reports*, 1924, lxxiv, p. 109. [2] SYMONDS, C. P., and OGILVIE, W. H., "A Case of Localized Suppurative Meningitis over the Motor Cortex," *Lancet*, 1922, ii, p. 272; and *Proc. Roy. Soc. Med.*, 1922, xv (Sect. Otol.), p. 39. [3] ADSON, A. W., "Pseudo-brain Abscess," *Surgical Clinics of North America*, 1924, iv, p. 503.

Discussion.—Dr. DAN MCKENZIE (President) said that Dr. Symonds had raised many points of novelty and interest. He (the speaker) had always particularly emphasized the points referred to in connexion with cerebellar abscess. It was usually stated that cerebellar abscess was more frequently missed than was temporo-sphenoidal abscess. If true, this seemed difficult to account for; he (Dr. McKenzie) wondered whether the point about nystagmus might not be one of the reasons. The books stated that in quite a large percentage of cases of cerebellar abscess no nystagmus was noticed. In some patients nystagmus was difficult to detect, but if the attention of a stuporose patient could be so directed that his eyes were turned towards the side of the lesion, a slow return to the neutral position would be noted, i.e., the slow component of the nystagmus was present, but not the quick one, because the stuporose condition prevented the operation of the higher nerve centres.

Dr. Symonds' reference to encephalitis was interesting, because cases were often seen in which there were symptoms apparently of brain abscess, which however was not found on exploration. Later on such patients nevertheless recovered. He (the speaker) had been called to see a boy who had a discharge from the ear, accompanied by headache and pain, and he had operated on the mastoid; he did not feel justified at that time in exploring the brain. Two days later, however, it was insisted that he should do so. The patient was semi-comatose, and the temporo-sphenoidal lobe was explored but with a negative result. The boy recovered.

There had recently been cases of encephalitis lethargica in which the symptoms had pointed to brain abscess. That diagnosis seemed to be confirmed if there was an ear discharge. With ordinary care there was no fear of harm from exploration of the brain.

In testing the integrity of the naming centre, Dr. Symonds had suggested giving many test objects. He (the speaker) had been accustomed to test with only two or three common articles, but apparently there was safety in a greater number. These points seemed trivial perhaps, but it was the summation of apparently trivial points that might make all the difference in a diagnosis.

Sir JAMES DUNDAS-GRANT said that Dr. Symonds' insistence on the signs of superficial abscess was a valuable point; he (Sir James) could look back on some puzzling cases and he now realized that they had been of that nature.

Mr. F. W. WATKYN-THOMAS said he had had a patient in whom for a short time, there had been pure motor aphasia, with a lesion of Broca's area on the left side. In this case nasal polypi had been removed, and the patient had come to the hospital having a fistula of the right frontal sinus and a superficial abscess. The right frontal sinus was opened and drained. The next day the patient had seemed well, but that night it was noticed that he "was talking nonsense." On the following morning he could say a few words, but they were unintelligible and disconnected. He could understand what was said to him, and could carry out suggested movements. There was right facial weakness, the tongue was protruded to the right, and

there was a double Babinski's reflex. Lumbar puncture was performed, and two hours later a right brachial monoplegia developed and the patient was quite unable to speak. He could still turn in bed, make suggested movements, and so on. The arm area on the left side of the motor cortex was exposed, and a loculated subarachnoid collection of purulent cerebro-spinal fluid was found. Both frontal sinuses were widely opened. The left sinus was full of polypi, but there was no perforation of the inner wall. The patient died the following day. No track of the infection was found at the post-mortem examination, but there was a well-defined depression over the anterior portion of the left sylvian fissure, the foot of the rolandic area, and the third frontal convolution. He (Mr. Watkyn-Thomas) had seen several cases of injury of the area of Broca on the left side, but this was the only one in which aphasia had been present.

In none of the cases shown that day was there any evidence of crossed deafness. This fact was important, as it seemed to throw doubt on the existence of a contralateral hearing centre in the temporal lobe. In none of these cases, further, was there any sign of a "pure word deafness." In fact their evidence was against the existence of verbal images. Chatelin and de Martel had suggested the existence of a centre for stereognosis at the foot of the ascending parietal, but the evidence was, unfortunately, weak. If any reliance could be placed on it, such a sign would be valuable for localization.

Sir WILLIAM MILLIGAN said that according to statistics, temporo-sphenoidal abscess was twice as common as cerebellar abscess, but his own experience was just the opposite. One recalled earlier and less accurate days, when the diagnosis of "brain abscess" was made without any precise idea as to its location in the brain. Many surgeons operated on the temporo-sphenoidal lobe because statistics showed that to be the most likely site for abscess. A few years ago little was known about nystagmus, and for that reason cerebellar abscess was frequently missed.

With regard to the withdrawal and examination of cerebro-spinal fluid, in every case which he (the speaker) had seen in which there had been leakage into the ventricles, and an almost maniacal mental condition had developed, accompanied by a rise of temperature.

Did Dr. Symonds think that non-suppurative encephalitis was due to the effects of a circumscribed serous meningitis? Symptoms of pressure existed to such an extent as to lead to the idea that an abscess was present. He (Sir William) suggested that these symptoms were due to a localized condition and that there had been time for adhesions to form in the infected area, so that as a result of plastic inflammation the membranes became soldered together. Two years ago he (the speaker) had had a case in which he had the opportunity of watching developments. He operated on a young man who had developed a condition of pseudo-brain abscess. He became quite unconscious and passed both fæces and urine in bed. There were no symptoms enabling one to say definitely that he had a brain abscess, but as his condition was so grave, he (the speaker) explored the temporo-sphenoidal lobe. A good recovery ensued, and he had followed the case up with great interest ever since. It seemed to have been superficial damage, of inflammatory nature, of questionable origin, and he would like to hear what Dr. Symonds thought was the probable course of events.

Mr. G. J. JENKINS said that some years ago the late Mr. Hunter Tod had described a series of cases in which there was softening of brain, and in which at the operation he had been able to distinguish healthy brain substance from the infected area.

Dr. C. P. SYMONDS (in reply) said that sometimes cerebellar abscess caused him more difficulty in diagnosis than did temporo-sphenoidal, especially when labyrinthitis and cerebellar abscess co-existed. In the present week he saw a case in which the symptoms might be accounted for by labyrinthine irritation, and it was only discovered at the post-mortem examination that the patient had a cerebellar abscess as well. Another difficulty was that with the variety of cerebellar abscess to which Mr. Jenkins had referred, situated on the antero-inferior surface of the cerebellum, close to the jugular foramen, which might give very few localizing signs.

Mr. Watkyn-Thomas's case was interesting as a good example of superficial abscess, distant from the focus of infection but with a clue as to the track.

There was more risk from lumbar puncture in cerebellar abscess than in temporal abscess. If there was a serious increase of intracranial pressure with drowsiness and swelling of the optic discs, lumbar puncture was contra-indicated. The amount of fluid removed in a

case should be small, 2 c.c. or 3 c.c. was sufficient. Because of the risk, he (Dr. Symonds) would not perform lumbar puncture in any case in which the diagnosis could be settled without it. That procedure should be reserved for cases in which there was doubt; in such cases it might afford just the information one was seeking.

In none of his (the speaker's) three cases of non-suppurative encephalitis had a leucocyte count been made, but in Adson's three cases a polymorphonuclear leucocytosis had been found such as would be expected in cerebral abscess, and therefore it was not a differentiating point.

Sir William Milligan had said that the symptoms in cases of so-called pseudo-brain abscess might be due to serous meningitis, but what he (the speaker) thought was that a collection of fluid on the surface sufficiently large to cause those symptoms must also be sufficiently large to be readily detectable at operation; and in the two cases of his own which were operated upon, and in Adson's cases, no such collection had been found. It took a large collection of fluid on the brain to cause aphasia, extensor plantar response on the opposite side and swelling of the optic discs. Further, in one case there had been involvement of the optic radiation, which could not have been caused by a superficial lesion. That was why he (Dr. Symonds) suggested the alternative explanation that these were cases of non-suppurative inflammation, something which never reached a breaking-down stage. He did not feel satisfied about this as a pathological proposition, because it did not take place in the ordinary way within the body. But it occurred in the skin, and an analogous instance could be found in amœbic hepatitis which often cleared up without any evacuation of pus.

Care should be taken in following up these cases. A patient might have a small brain abscess with a surrounding area of encephalitis, which might cause symptoms and signs, and as the abscess became more shut off the physical signs would diminish, though the abscess remained. Such an abscess might cause further symptoms, even after an interval of three years, as in a gunshot wound case of his own.

He had purposely not dealt with the atypical varieties of brain abscess, partly because he had not had much experience of them. He had seen one case of anterior temporal abscess with uncinate attacks, and two or three cases in which an abscess of the antero-inferior surface of the cerebellum was present without any of the inco-ordination of the limbs usually found in a deeply situated abscess of the lateral lobe.

ILLUSTRATIVE CASES.

Cerebellar Abscess.

By R. J. Cann, L.R.C.P.Lond., M.R.C.S.Eng. (introduced by Mr. T. B. Layton).

R. A., MALE, aged 20.

In 1924, discharge from left ear. A polyp was removed—intermittent discharge afterwards.

Seen again November 4, 1926. Recurrence of aural discharge for one week, following a cold. During subsequent week complained of severe diffuse headache, frequent vomiting—became visibly thinner.

November 11, 1926.—Aural discharge profuse; drumhead obscured by granulation; no mastoid tenderness; slight facial weakness left side; no nystagmus; suspicion of paralysis of left external rectus muscle; reflexes normal; pointing error with left arm observed once, but not repeated; one observer thought nose-finger-nose test deficient on left side; headache now referred to occipital region; some rigidity of neck; temperature 100° F.

Same evening cortical mastoid operation by Mr. T. B. Layton; bone dense; pus in mastoid antrum; granulations on dura over transverse sinus; dura of middle and posterior fossæ exposed.

November 12, 1926; afternoon.—Patient comfortable, answered questions readily. Temperature, 98·6° F.; pulse, 70; left-sided facial weakness still present: left pupil smaller than right, both reacting to light; coarse horizontal changing nystagmus, more marked to left than right; knee- and ankle-jerks exaggerated; Kernig's sign negative; rigidity of neck unchanged; inco-ordination of left arm shown by nose-finger-nose test; diadokokinesis positive for left arm.

Later same afternoon patient suddenly complained of intense headache and required morphia to ease him.

November 13, 1926; morning.—Patient could not be roused. Reflexes as on previous day; pulse 56 to 60. Seen by Dr. Symonds, who diagnosed left-sided cerebellar abscess and advised immediate operation. Pulse gradually rose, and immediately before operation was 90 and irregular. At operation (R.J.C.) a large abscess was found in left cerebellar lobe with 1 oz. of serous foul-smelling pus, cerebro-spinal fluid hazy. Patient made a good recovery and was discharged on December 22, 1926.

Left-sided Temporo-sphenoidal Abscess with Aphasia.

By W. M. Mollison, M.Ch.

For account of case see E. H. Richards, in *Guy's Hospital Reports*, 1924, lxxiv, 109.

Superficial Abscess of the Brain.

By W. H. Ogilvie, M.Ch. (introduced by Mr. T. B. Layton).

See *Proceedings*, 1922, vol. xv (Sect. Otol.), 39.

After-history.—For the first two months after discharge from hospital there was some mental confusion, and she was very emotional. Since that time she has had no trouble. She has had no fits or fainting attacks. In spite of long absence she was in the top class at school. At the age of 14 she was hit over the skull defect by a cricket ball, and was unconscious for one hour. She now earns a living by making hats.

Present Condition.—General health good. There is an extensive cranial defect, but no tenseness or protrusion of cranial contents. Deafness in left ear.

Slight facial weakness on right side. Motor power and reflexes in limbs equal on both sides. Grip of right hand as good as that of left. No astereognosis on right side, movements a little more deliberate than those of the left hand. No sensory impairment.

Superficial Abscess of the Brain.

By C. Gill-Carey, F.R.C.S.Ed.

Girl, aged 9. Admitted to hospital April 8, 1926, with acute mastoiditis (left). Operation same day; pus in mastoid cells; dura mater of middle fossa injured but not perforated by a spicule of bone.

Convalescence uneventful until April 29, when, at 3 a.m., she became unconscious and foamed at the mouth; and coarse tremors developed on the whole of the right side of the body. Consciousness regained at 8 a.m. Movements of the right side were then localized to the leg.

Operation same day. Dura opened at the site of injury and pus found about $\frac{1}{4}$ in. from the surface.

Temperature 103° F.: pulse 160. Cerebro-spinal fluid: polymorphonuclears 80 per cent.; protein 0·8 per cent.; sugar normal.

After this operation aphasia and right hemiparesis developed. Neurological examinations by Dr. C. P. Symonds:—

May 2, 1926.—"Aphasia; right hemiparesis; bilateral extensor plantar reflexes."

May 11, 1926.—"Mentally alert; severe aphasia of temporal lobe type. Optic discs: right, doubtful; left, slight swelling. Right hemiparesis with apraxia; bilateral extensor plantar reflexes, not with stiffness. Suspicion of pus still in left temporal lobe."

Sinus forceps were pushed into the temporal lobe and another pocket of pus was opened. Slow but steady improvement followed.

Localized Non-suppurative Encephalitis.

By W. M. Mollison, M.Ch.

Boy, aged 10. Admitted to hospital September 11, 1926. Pain in and behind right ear following acute otitis media of one week's duration. September 12: temperature 100° F.; pain persistent with much discharge from the ear. Operation on same day—pus found in the mastoid cells and a small collection compressing the lateral sinus which was, however, not thrombosed. The dura in middle fossa exposed, but not incised—wound drained. After operation temperature in the evening was between 99° F. and 105° F., pulse ranging from 90-120. He was noted to be odd, apathetic (not speaking unless spoken to) and taking no interest in his toys. On nineteenth day after operation doubtful extensor response obtained from left foot.

About this time began to complain of right frontal headache, chiefly on waking in morning; occasionally associated with nausea. First seen by Dr. C. P. Symonds on October 7, i.e., twenty-five days after operation. A definite extensor plantar response on left with diminished abdominal reflexes on same side was found. Right temporal abscess was diagnosed and an operation performed on same day—the dura being opened and brain explored, no abscess being discovered. Lumbar puncture at time of operation produced clear fluid apparently under increased pressure, containing no increase of cells or protein. Following this operation condition remained much the same. Temperature and pulse-rate still high, a good deal of complaint of headache but otherwise apathy was the most noticeable feature. Six days after second operation the physical signs were unchanged and it was suggested that it might be a case of non-suppurative encephalitis. It was decided, therefore, not to undertake any further operation for the time being, and to give full doses of hexamine. On October 16 (five weeks from the date of admission) patient began to improve, temperature and pulse fell and headache was less; he took a natural interest in his surroundings. On October 29 he seemed normal and an extensor response from left foot could no longer be obtained. Has since remained well. Possibly an abscess may be present but the provisional diagnosis of non-suppurative encephalitis will probably prove correct.

Right-sided Temporo-sphenoidal Abscess without Localizing Signs.

By T. H. Just, F.R.C.S.

J. S., male, aged 6. Was admitted to hospital on November 3, 1924, with a history of discharge from the right ear for two months, slight headache for two weeks, and having lost flesh. On admission the boy looked ill, and was a little drowsy. The right ear was discharging freely, the drum perforated and granulations in the tympanum. There was slight tenderness over the right mastoid, no œdema

or swelling. Temperature 97·6° F. Pulse 100. Respirations 24. The boy had not vomited; there was no nystagmus. Reflexes normal.

November 11, 1924.—Mastoid tenderness has gone. Still a little apathetic. Discharge from ear copious. No other physical signs. Temperature 97·4° F. Pulse 98. Respirations 24.

Diagnosis:—Mastoiditis, extradural abscess.

Operation:—Mastoid opened. Bone acellular, cholesteatoma found in antrum. Sinus exposed, healthy. Extradural pus, with offensive smell. Dura mater over middle fossa covered with granulations. Dura mater pulsating normally. Radical mastoid operation performed. In view of the offensive nature of the extradural pus, the dura mater of the middle fossa was incised, and the temporo-sphenoidal lobe explored. A large cortical temporo-sphenoidal abscess, containing 2 oz. of pus, was opened. This was washed out with saline solution, and drained with a rubber tube.

Lumbar puncture performed at the time of operation: Fluid under some pressure; 42 cells per c.mm.; culture sterile.

Pus from abscess:—Streptococci, on film and culture.

Uninterrupted recovery. Wound healed well. General condition good; cheerful and well, but is a little more excitable than before the operation. Discharged December 12, 1924.

Left Cerebellar Abscess.

By T. H. Just, F.R.C.S.

I. S., FEMALE, aged 5, was admitted to hospital on June 11, 1926. Discharge from the left ear. "Low spirits," losing weight, and some headache. Had had acute otitis media May 28, 1926; left drum incised. Had made a good recovery; but discharge had recommenced.

On admission.—Temperature 97·8° F.; pulse 92; respirations 18. Lies quietly on back. Occasional sucking of teeth. Flexion at hips and knees. Answered rationally. No cry.

Tongue moist, furred. Slight otorrhœa, left. *Eyes*, pupils dilated, symmetrical. Reaction to light; ? reaction to accommodation. Ocular movements good; no strabismus. *Fundi*—edges of discs blurred. *Reflexes* present, and equal. *Upper limbs* normal, reflexes normal; no dysdiadokokinesis. *Lower limbs*—flexion at hip and knee, variable. Kernig's sign present. Knee-jerks not obtained. No ankle clonus.

June 16, 1926.—*Lumbar puncture*.—Fluid under slight pressure; cells 136, nearly all lymphocytes; globulin normal; reducing substance normal; albumin, a slight trace.

Blood-count.—Red blood-cells 5,900,000; white blood-cells 14,400; hæmoglobin 80 per cent.; colour index 0·7.

Eyes.—Well-marked papillœdema, right and left, of the type that is associated with cerebral tumour. (Report by Mr. Foster Moore.)

Operation.—Mastoid opened up. Pus found. Dura mater over middle and posterior fossæ exposed widely. Pus over posterior fossa of dura mater, which was injected and tense, though pulsating. Posterior fossa dura incised and cerebellum explored when a large cortical cerebellar abscess was immediately opened. The abscess cavity was washed out with saline and tube drained.

Pus from abscess.—Film—short-chained Gram-positive cocci. Culture—sterile.

June 17, 1926, 10 a.m.—General condition good. Complete blindness. Pupils widely dilated. 3 p.m., vision returned.

June 20, 1926.—General condition good. No headache. Still transient attacks of blindness lasting from a few minutes to two hours. Power and co-ordination of limbs good.

June 22, 1926.—Reflexes as before. Papilloedema less marked (R. F. M.). Recovery very good. General condition excellent. Patient discharged from hospital, July 3, 1926.

August 20, 1926.—Visual acuity very defective: secondary optic atrophy of both discs more marked in the left. There is now no active change. (R. F. M.)

October 8, 1926.—Visual acuity: left eye, $\frac{6}{36}$; right eye less than $\frac{6}{60}$. The general condition is excellent. The granulating wound behind the ear has practically healed.

Right Cerebellar Abscess.

By G. W. Dawson, F.R.C.S.I.

D. B., female, aged 25.

Seen October, 1918.—Had had right otorrhœa all her life and then had slight tenderness and swelling of the mastoid, with pain right side of head. A few days previously had had facial paralysis, and giddiness. Temperature 99·8° F.; pulse 100. For the previous twelve months she had "felt seedy," with headache and loss of appetite. She had lost weight and colour and was easily fatigued. Memory not affected.

October 8.—Radical mastoid operation: bone dense and acellular; pus in antrum. For two days she felt better. Temperature dropped to normal and pulse to 60, but on October 10 the temperature again rose to 100° F., where it remained till October 20, the pulse remaining at 60.

October 18.—The headache referred to right occiput became worse and on the 19th she vomited, and spontaneous nystagmus to the affected side was noticed. Fundus examined by house surgeon: slight papillitis.

October 20.—Screaming with pain she lay on sound side curled up in bed. Tongue dry and brown; pulse (64) small and weak; no weakness of limbs; reflexes normal; intellect perfectly clear, answered questions quickly. Cerebellar abscess was diagnosed.

Operation.—Mastoid re-opened: no track found. Attempt made to open cranial cavity in front of sinus, but space was too limited. Trephined behind sinus, and passed knife in several directions into cerebellum.

I almost abandoned the operation when I found a large quantity of fœtid pus, which appeared to be situated low down and anteriorly. The amount appeared so large that one wondered how it could be missed. An iodoform gauze plug was introduced and changed every day. The nystagmus disappeared in a few days. The temperature dropped to 99° F. in four days, and in six days she was free from pain and was allowed out of bed on November 12. Her ill health and progressive loss of weight and colour during the previous twelve months seem to indicate the presence of a chronic abscess.

Right Temporo-sphenoidal Abscess.

By G. W. Dawson, F.R.C.S.I.

F. N., MALE, aged 21. Right otorrhœa three years. Occasional pain about December 10, 1926; became ill with pain in ear; temperature 100° F. Recovered in a few days.

December 29, 1926.—Became unconscious at night and was brought to the hospital.

December 30, 1926.—Coma from which he could not be roused. No paresis—patellar reflexes absent; plantar reflexes active; König's sign absent. Fundus examined next day—normal. Temperature 101° F.; pulse 72. Radical mastoid—cholesteatoma—sinus found in region of antrum. Probe passed upwards 2½ in., not much discharge; the opening was slightly enlarged and an iodoform gauze plug inserted. On changing this next day there was a profuse discharge of fœtid pus and brain matter. Tube inserted but it had to be changed twice daily as it appeared to dam up the discharge. He recovered consciousness after the operation but was violent for several nights.

January 5, 1927.—Except for a slight headache is very well, but much discharge persists.

January 19.—Very well, discharge still considerable.

The temperature has been normal since operation and the pulse round about 60 except on two occasions when it dropped to 54.

Left Temporo-sphenoidal Abscess.

By SYDNEY SCOTT, M.S.

S. P. C., MALE, aged 44. Referred by Dr. Hinds Howell.

History.—Three weeks ago: "influenza," left otalgia, no discharge. In bed three days, then out and about. Two weeks ago: back to bed, headache, chiefly left side. Vomiting once or twice daily. Had a shivering attack. Some mental wandering. Left otalgia, with discharge, began two days after return to bed; discharge has continued since. One week ago became drowsy and somewhat incoherent; very severe headache, mainly left temporal. Marked paraphasia noticed before admission.

Condition on Examination: September 28, 1924.—Patient semi-comatose, talks incoherently, yawns repeatedly, tongue dry and furred. Left otitis media. Pus in meatus. Slight hemiplegia (right). Temperature 97·6° F.; pulse 52; respiration 20. *Cranial Nerves*: Right seventh, weakness; others normal. *Reflexes*: Right biceps, supinator and abdominal muscles, reflexes not obtained. Slight weakness of right upper extremity. Right knee-jerk could not be tested owing to ankylosed knee. Right ankle-jerk slightly diminished. No flexor plantar response, Left side normal. No sensory changes. *Eyes*: Normal.

Diagnosis.—Left acute suppurative otitis media with left temporo-sphenoidal abscess.

Operation: September 28, 1924.—Left ear: Schwartze's operation; cellular mastoid; pus in antrum and cells. Tegmen antri removed; dura mater bulging under tension; feeble pulsation. Exposed more widely. Brain explored: Abscess found ¼ in. from the surface; ½ oz. slightly offensive pus evacuated. Drained with six small tubes. Wound packed open.

Bacteriology of Pus.—Pure growth of streptococci.

Subsequent Progress: November 4, 1924.—Discharged cured; ear dry; mentality and speech apparently normal.

Left Temporo-sphenoidal Abscess.

By SYDNEY SCOTT, M.S.

L. B., AGED 22. First complained of deafness and discharge from the left ear in May, 1926: Headaches, left frontal.

Owing to repeated attacks of headache (left frontal) she was admitted to hospital on December, 30, 1926, and the mastoid was opened and drained. The antrum

54 Scott—Dundas-Grant: *Temporo-sphenoidal Abscess*

was comparatively small but was full of pus. She seemed much better after this but was kept under observation. Five days later she had an attack of feverishness, temperature rising from normal to 103° F., and appeared to have an attack of "influenza," the feverishness gradually passing off and the temperature becoming normal in three days.

January 8, 1927.—Blood-count: White cells = 16,400. January 9, 1927.—Blood-count: White cells = 16,500.

After the feverishness ceased the pulse became abnormally slow, on two occasions 56 and 48 respectively—but was most of the time between 70 and 80.

About January 8 when the temperature was normal the nurses noticed that she was irritable, and was asking why she should not sit up and wash herself, like other patients in the ward. She was annoyed with herself because she could not remember the names of various articles. She did not sleep well.

On January 10 I decided to explore the brain because she called a coat button "an envelope," and keys she called an "envelope," though she could read and spell and count accurately. She could distinguish any coins quickly and correctly, but a medallion puzzled her. There was no contraction of the visual fields.

The same night a large collection of pus was found in the left temporo-sphenoidal lobe immediately above the mastoid antrum, and the headaches have been relieved ever since.

Right Cerebellar Abscess.

By Sydney Scott, M.S.

S. B., girl, aged 12, complained of chronic discharge from right ear. For three days before admission to St. Bartholomew's Hospital on November 9, 1924, she had had earache, giddiness and was sick. Right mastoid region œdematous and tender; offensive purulent discharge from tympanum. Schwartze's operation. Extradural abscess discovered. Sigmoid sinus opened, clot removed. Owing to rigors, occurring November 14 and 15, 1924, internal jugular vein ligatured; rigors and pyrexia then ceased.

November 20 to 23, 1924.—Headache.

November 26, 1924.—Nystagmus to the right, ataxia hypotonia; dysmetria and dysdiadokokinesis on right side.

Cerebellar abscess opened; 2 drachms of foul pus evacuated (hæmolytic streptococcus and *Bacillus proteus* isolated).

February 4, 1925. Radical conversion and plastic operation.

February 11, 1925.—All signs of cerebellar lesion had disappeared. Patient has remained well since.

Temporo-sphenoidal Abscess.

By Sir James Dundas-Grant, K.B.E., M.D.

[The complete history is to be found in the *Transactions of the Otological Society* (February 1902) and in the *Journal of Laryngology* (March 1902)].

The favourable factor in this case was probably the short duration of the abscess and the absence of anything in the way of a rigid capsule to interfere with the obliteration. No plastic measures were taken, but the state of the lining of the walls of the mastoid "operation-cavity," as shown before the Section in March 1924,[1] was quite perfect.

[1] *Proceedings*, 1924, xii (Sect. Otol.), 73.

Cerebellar Abscess Secondary to Thrombo-phlebitis of the Bulb of the Jugular Vein.

By Sir JAMES DUNDAS-GRANT, K.B.E., M.D.

THE details of this case, in regard to the diagnosis and treatment of the thrombo-phlebitis of the jugular bulb, were published in the *Journal of Laryngology* (March, 1906).

The patient, aged 27 at the time of the operation, served in the late war and was wounded in the upper part of the right arm, so that the past pointing tests are not practicable.

Large Temporo-sphenoidal Abscess following Injury: no Localizing Signs except Homolateral Papillœdema.

By E. BROUGHTON BARNES, F.R.C.S.Ed.

V. W., MALE, aged 12. Seven years ago radical operation on the right mastoid in Italy.

June 5, 1926.—Knocked down by a car. Admitted to hospital suffering from concussion. Detained eight days. Discharged as well.

Mother reported that on the thirteenth or fourteenth day after the injury he vomited and had rigors several times.

First seen by exhibitor on the fifteenth day after the injury. Pulse 46. Temperature 96·6° F. Did not look ill. Complained of severe frontal headache at times. Very drowsy. There was a wound about 2 in. long, vertical, immediately in front of the right pinna. It appeared to be quite superficial and was slightly inflamed. Unsatisfactory radical cavity (right) with a considerable amount of foul discharge appearing to come from the roof of the mastoid part of the cavity. Chronic suppurative otitis media with very foul scanty discharge (left).

Very slight papillœdema of the right disc. Grips equal. All reflexes normal. Mentality slow.

Operation same night. Mastoid scar excised. Incision carried forward above the pinna. A pus-filled cavity with smooth walls about the size and shape of a robin's egg formed the roof of the radical cavity and extended into root of the zygoma. A fracture extended from the mastoid cavity through this abscess, forward and upward, into the squamous part of the temporal bone. There was about 2 drm. of extradural pus beneath the root of the zygoma. A detached spicule of bone had not penetrated the dura mater.

The radical cavity was cleaned out, and many unopened infected cells were removed posteriorly and towards the tip. The root of the zygoma and the large abscess cavity were removed, and an area of unhealthy dura was exposed as far forward as the anterior wall of the meatus. The temporo-sphenoidal lobe was explored forward and a large quantity, estimated at about 2 oz., of thick, yellow pus, was evacuated. The cavity appeared to occupy the whole of the front of the temporo-sphenoidal lobe. Drainage through rubber tube stitched to the dura.

Cavity contracted slowly; tube removed on nineteenth day. Wound closed on twenty-seventh day; fresh meatal flap cut.

Recovery uneventful except for slight secondary hæmorrhages from the anterior scalp wound into the cavity on the fifth and seventh days. On the second occasion the wound was re-opened, and the vessel was successfully ligatured. During convalescence the boy was bright and cheerful, and free from headache. Never any trace of paresis or alteration of reflexes. He was discharged on the forty-first day, August 6, 1926, and has been perfectly well since.

Left Temporo-sphenoidal Abscess.

By E. Broughton Barnes, F.R.C.S.Ed.

W. S., MALE, aged 29. Admitted to hospital September 18, 1925. Looked very ill. Temperature 103° F. Pulse ranged between 70 and 100. Moaned continually—held forehead—talked nonsense—answered when spoken to—restless. The whole interior of the nose was brilliantly red and dry. Right ear: subacute catarrhal otitis media. Left ear: tympanum full of œdematous granulations. Very little pus. No œdema of the posterior bony wall. No tenderness or œdema over the mastoid. Pupils equal. Very irritable, shouts when touched. Neck rigid. Kernig's sign present both sides. Knee-jerks exaggerated. Definite ankle clonus both sides. Babinski's sign negative. Discs normal. Lumbar puncture: fluid (loaded with blood) under greatly increased pressure.

Radical mastoid operation. Cholesteatoma cavity, extending upward from the attic, had exposed dura of the middle fossa. Wound packed open. Patient apparently *in extremis*. Thirty-six hours later, lumbar puncture, fluid under raised pressure, contained polymorphonuclear leucocytes: culture sterile. Sixty hours later all meningeal symptoms had disappeared. Ophthalmic surgeon's report: No neuritis—discs hyperæmic only—no exudate—no hæmorrhages. Still intense headache localized to left frontal region. Mentality appeared slow. Pulse 68. Temporo-sphenoidal lobe explored—abscess containing about 1 oz. of pus evacuated from posterior part of lobe. Tube removed on eighth day. Recovery uneventful. Occasional headache for four months: none since January, 1926. Has been in full work since.

Loculated Cerebral Abscess.

By W. M. Mollison, M.Ch.

L. R., GIRL, aged 13, admitted to Guy's Hospital on October 18, 1926. Had suffered from earache and otorrhœa "for years."

Seventeen days before admission to hospital had had severe and progressive frontal headache and pain over the right eye.

On admission: Somewhat lethargic, pain over right eye, and headache; knee-jerks and ankle-jerks sluggish, right extensor plantar reflex; Kernig's sign positive; optic

discs normal; pulse-rate 53. The right tympanic membrane showed a large perforation discharging some foul pus; no tenderness over the mastoid process.

Operation.—Foul pus emitted under pressure found in small cells in the mastoid and in the antrum granulations on the lateral sinus. Lumbar puncture showed the fluid under increased pressure.

Some temporary improvement followed; further operation undertaken in order to explore the middle fossa; pus drained from temporo-sphenoidal lobe; in spite of this drainage pulse remained at 56 and headache was unrelieved. Another operation: on exploring the previous opening no more pus was found; a finger was then introduced into the brain and a smooth, slightly tense swelling felt on the posterior and inner wall of the abscess cavity; a forceps thrust into this swelling released a large amount of pus; patient made a slow recovery. The pus contained *Staphylococcus aureus* and *Bacillus xerosis*.

Two Cases of Cerebellar Abscess: Previously Reported.[1]

By SYDNEY SCOTT, M.S.

(I) Cerebellar abscess; sudden coma and apnœa; recovery after operation during artificial respiration.

(II) Cerebellar abscess five weeks after onset of acute otitis media, right side.

Left Temporo-sphenoidal Abscess: Amnesia for Names of Objects: Previously Reported.[2]

By SYDNEY SCOTT, M.S.

Left Temporo-sphenoidal Abscess Opened Sixteen Days after the Onset of Acute Otitis Media. Outstanding Symptom Auditory Amnesia ("Name-Amnesia").

By SYDNEY SCOTT, M.S.

R. H. G., MALE, aged 36, caught severe "cold in head" on July 2, 1923, followed by acute earache and spontaneous otorrhœa on July 5. Otorrhœa and slight pyrexia (100-101° F.) lasted seven days, and during the second week he was free from all symptoms, but in spite of this he became physically and mentally inert, and remained in bed showing no wish to get up. He lacked initiative, and used incorrect words in conversation. For instance he called a glass stopper "a bung," a pencil he called "a calculator," a tuning-fork was also "a calculator," a comb was "for the

[1] See *Proceedings*, 1923, xvi (Sect. Otol.), 56, 57.

[2] *Ibid.*, 55, 56.

58 Scott: *Left Temporo-sphenoidal Abscess*

household," a brush was also " for the household "—instead of for the " hair of the head." The neurological reflexes were all normal. He was not sick, and had no headache even after sitting up and bending the head over the knees while the lower extremities were extended.

The diagnosis of a temporo-sphenoidal abscess depended solely on the mental inertia, and auditory paraphasia. An exploratory operation was carried out, the abscess discovered in the cerebrum immediately above the middle ear and drained. The mucosa of the antrum was slightly swollen, but there was no pus in the tympanum.

After the operation, he was found to have right-sided homonymous hemianopia without involvement of the macula (Sir William Lister). This hemianopia still persists—over three and a half years since the operation,. but he is a highly intelligent, capable, active man, having adapted himself to the narrowed visual field. He was recently re-insured as a first-class life.

Section of Pathology.

President—Dr. JAMES MCINTOSH.

The Part played by Injury and Repair in the Development of Cancer, with some Remarks on the Growth of Experimental Cancers.

By Professor H. T. DEELMAN (Groningen).

CAN we demonstrate experimentally the reason for the very localized starting point of a tumour, although a great number of epithelial cells had been in contact during days and weeks with the cancer-producing agent? (tar in the case of the experimental tar cancer).

Mice were tarred in the usual way till some of the animals showed early papillomata, which, when seen under the microscope, proved to be definitely localized epithelial hypertrophies. If tarring is continued at this stage of the experiment the skin of the mice tends to form numerous tumours in a very short time. On the other hand, if tarring is discontinued, only a very small number of tumours will appear. At this stage of the experiment we therefore stopped tarring. A few days afterwards we made incisions in the skin, taking care to make these incisions at places where no papillomata had appeared. These wounds very soon assume an oval shape and heal in the normal time. At first there is formed a layer of fibrin and dead cells, and under this material we very soon found the first indications of healing in the shape of new epithelial cells. In the majority of cases we found that new tumour-processes had formed in the healed or partly healed wounds. In some cases there were only two or three tumours in one scar, in others there were many more. I never observed that the whole scar was involved in the tumour process. Sometimes the tumour growths were so numerous that the scar could be localized with the greatest ease. I have just mentioned that the tumours appeared in some cases after the scar had healed and that the new epithelial cells—that is to say, cells never themselves in contact with the tar—produced in the course of these experiments primary growths. We know that there is a relation between tar and tumour formation; we now learn from these experiments that the liability to tumour formation can be transferred to daughter and grand-daughter cells without the cells showing any biological change microscopically. These tumours, which we produced just at the place where we wanted to see them, were very malignant and sometimes the whole scar and the surrounding tissue were very soon replaced by a typical cancer growth. We have thus an opportunity of obtaining a clearer insight into the local origin of tumours in a skin area over which all the cells have been in contact with the cancer-producing material. The animals every now and then receive minute wounds. These wounds may be so small that they escape attention. They heal as a rule very quickly and we may suppose that some of them will produce a tumour in the same way as was shown in our experiments. In a very short time these tumours may become larger than the little (often almost invisible) scars from which they originated. These experiments may have a general significance as regards the local origin of a tumour.

Some other experiments throw light on the much discussed question of the manner of extension of a tumour. When we study the earliest beginning of a tumour there is seen to be a local hypertrophic growth. Somewhat later the

20 Deelman: *Part Played by Injury in Development of Cancer*

surrounding cells hypertrophy as well. This process is extended until at length there is present on the skin a hypertrophic proliferation, generally in the form of a papilloma. The way in which such a papilloma grows seems to indicate an influence of the central parts of the papilloma on the surrounding cells of the living organism, with the result that these also hypertrophy. At a later stage the central parts of the papilloma become transformed into cancer cells and we have noticed that the cancer cells influence the surrounding hypertrophied cells exactly in the same way as the hypertrophied cells have previously influenced the surrounding normal cells.

These observations on the growth of young tumours, as was demonstrated at the Cancer Congress held at Strasbourg in 1924, confirm the original view maintained by Virchow. It is of great importance to show in another way the influence of tumour cells upon the surrounding living cells of the organism.

At the beginning of this paper I demonstrated that under certain circumstances young (regenerating) epithelial cells are very susceptible to tumour formation. On the strength of that observation I carried out the following experiment on the mice:—

Tar was applied to the epilated skin until a few small papillomata appeared; we then stopped applying the tar. A few days later we made two small scarifications, one which was in contact with the tumour, the other at some distance, for the purpose of control. We marked the position of these wounds by rubbing in Chinese ink and after two or three days the wounds had become healed. Only a small black line under the newly-formed epithelium showed us the place at which the fine, small scalpel had cut through the upper part of the skin. The minute healing wound which either touched the tumour, or very nearly did so, showed some interesting changes. The whole mass of new epithelial cells in this wound has become transformed into hypertrophic growth. We have never seen such extensive changes in scars not in contact with a tumour.

There must be an influence exercised by the tumour cells on the cells of the scar. As these cells are very liable to become neoplastic, this influence may be clearly demonstrated. Just as we have seen in the instance of papilloma, the surrounding cells are predisposed to hypertrophic—and afterwards to real—tumour growth.

SUMMARY.

(1) Injury may exercise a direct influence on tumour genesis in an extensive area of epithelial cells that are susceptible to tumour formation.

(2) The results of former experiments have made it probable that in the earliest stages of tumour formation the peripheral cells may exercise an influence on the cells in the immediate neighbourhood. When we effect a contact between the youngest tumour cells and cells which we know are very susceptible to develop a tumour, we can then clearly see the direct influence of the tumour cells on the other cells of the organism.

It will be the purpose of further cancer study to obtain a better insight into these intercellular activities.

War Section.

President—Group Captain HENRY COOPER, D.S.O., R.A.F.

Diet and Disease in the Navy.

By Surgeon Commander C. H. DAWE, R.N.

IN dealing with the effects of any particular trade or calling on the health of the members of that community, we are at once faced with the difficulty of obtaining a satisfactory standard for comparison, such as the incidence of disease in the civil population. With the exception of tuberculosis, the Registrar-General's official figures are mortality rates. In the Services, we are more concerned with the case rates than mortality rates, and we have special populations with regard to selected men and age-groups. In England and Wales we have the male population, at each age-group, decreasing roughly with increasing ages, whereas, in the Navy with a population aged 15 to 45, we have, compared with the civil population, a high percentage of men aged 20 to 30, and a low percentage below 20, and above 30 years of age. With these difficulties, it becomes most practicable to compare the health of a service with what it was years ago, and to compare it with other services working under different conditions.

In the past forty years the general conditions of living in the Navy have vastly changed, the changes being probably more marked than in any other community.

Briefly stated, the changes consist of, (*a*) much improved diet, (*b*) more comfort, (*c*) less physical energy expended per man per diem, three factors which are intimately concerned in the nutrition and resistance of the body to infection, and in general metabolism.

In the Army it would appear that with men living ashore in barracks and married quarters fresh food has been more easily obtained than afloat, and as drilling in the open air is part of a soldier's daily work, the changes in nutrition and metabolism have probably not so greatly changed in the past forty years as in the Navy. Similarly, in the Air Force, we have a body of men living ashore and working in the open air mostly, factors which should result in a low sick rate.

In the early 'eighties, the Navy consisted of small steamships with small complements. At sea full use was made of sails, and masts and yards demanded much physical energy. Boat-pulling was a necessity, and the percentage of men employed below decks was small. Men spent a large part of their time in the open air and sun. Below decks, the cubic capacity was small and ventilation bad, stoves and steam fans being used to improve the air conditions.

The standard ration included 1 lb. of fresh meat and ½ lb. fresh vegetables when procurable. At sea, however, salt meat was the ration, salt pork and split peas every other day, salt beef with flour, suet and raisins one day, and preserved meat with rice and preserved potatoes on the other day. No flour was carried for bread making, so 1¼ lb. ships biscuit was issued daily at sea, soaked in water to make it soft and eatable. Oatmeal, sugar, tea, cocoa, and condiments completed the ration, which worked out at about 4,500 calories per diem. Bread of varying qualities was purchased locally. Rum and lime juice were available then as at the present time.

It should be noted that men had no option in this diet, i.e., they could not omit part of it and take up money in lieu. Canteens existed in the big ships only, so the majority of men lived practically on the standard ration, and I understand enjoyed healthy appetites. Cooking arrangements were primitive. An able seaman's pay was 1s. 8d. per day. It should also be remembered that commissions in those days lasted from three and a half to five and a half years, giving plenty of time for any ill-effects of the ration to show themselves. Considering the work done the calories were not excessive. There were plenty of fat and protein and much roughage in the carbohydrates, peas and raisins, but the diet was ill-balanced owing to the lack of fresh vegetable, and the food was monotonous.

According to McCarrison, the functional perfection of the gastro-intestinal tract is dependent largely on the adequate provision of vitamins, and salts in fresh vegetable foods. Lack of them produced impaired vitality of the tracts in animals, together with invasion of the mucous membrane and blood-vessels of the bowel by micro-organisms and ulceration of the duodenum and stomach.

Regarding the body as a transformer of energy, the digestive tract is responsible for the assimilation of most of the energy, and the skin gets rid of most of that energy in the form of heat. The integrity of these two great surfaces is therefore most important in the well-being of the body and depends primarily on nutrition.

TABLE I.—RATE PER 1,000 PER ANNUM.

	1882	1910	1923
Total sick rates	1,148	653	559
Injuries	240	140	96
Sickness only	908	513	463
Skin and cutaneous	272	76	62
Boils, ulcers, abscesses	80 per cent.	—	50 per cent.
Digestive system	150	100	75
Sore throat	25	50	30
Disease of stomach	25	10	10
Disease of intestines	60	30	20
Other diseases of the digestive system	41	10	15

In Table I are compared the sickness rates in the Navy forty-four years and sixteen years ago and the present time. It will be seen that in the 'eighties roughly one-third of the sickness, accidents excluded, was due to skin trouble, 80 per cent. of which were boils, ulcers and abscesses, and one-sixth of sickness due to digestive system troubles. That is, 50 per cent. of the sickness was due to skin and digestive tract troubles.

Dyspepsia, diarrhœa, colic, etc., were the most common forms of sickness. The other diseases of the digestive system were largely due to dysentery and liver abscesses. I suggest that in those days nutrition was at fault, and when the men were exposed to the heat and humidity of a warm climate, infection readily occurred, with lowered resistance of the skin and digestive tract. The entamœba readily gained a foothold and produced its sequelæ, causing the greatest number of deaths from diseases of the digestive system.

Comparing the figures of 1882 with those of 1923, the "sickness only" rate is halved, and roughly 60 per cent. of this fall is due to the improved rates in skin and digestive system. The "skin rate" has fallen from 272 per 1,000 to 62, and only 50 per cent. now is due to boils, ulcers and abscesses. Digestive system troubles are halved, but now new troubles, such as appendicitis and peptic ulcers have arisen.

At the beginning of the century ships were bigger, and sailing at sea a thing of the past. Engine-room complements and the speed of ships had increased and less time was spent at sea. Commissions were only for two or three years. Ship's complements

had increased, the number of men employed on the upper deck relatively decreased, and the amount of physical energy expended in the open air diminished. But there still remained for the majority of men a good deal of open-air life on deck, in boat pulling and sailing boats.

With the introduction of electric fans and steam heating, the air conditions in ships were generally improving. The pay was still 1s. 8d. per diem, but the comfort of the man afloat and ashore had much improved, and alcoholism and its sequelæ had decreased. The changes in the standard ration made the diet less monotonous and included one pound of vegetables instead of half a pound. Milk and jam were added, and it became possible to take up money in lieu of part of the ration, this enabling the men to purchase more freely from the canteens. With increasing time spent in harbour much more fresh food became available. Consequently, salt beef was abolished in 1905.

To-day a man takes up a reduced standard ration, plus a definite money allowance daily, or he is on the general messing system by which he is fed on the value of the standard ration and receives no money allowance. His pay as able seaman is 4s. a day, and he gets four good meals a day, excluding what he may buy at the canteen. It is, therefore, impossible to get the number of daily calories. With bakeries on board and cold storage, ship's biscuits and salt meat are no longer required.

On certain stations fresh food is not easy to obtain, but on the whole the naval man has a generous and varied diet.

The present-day ships are big, with large complements, and an ever-increasing percentage of men employed below deck in relatively sedentary occupations. The old masts-and-yards sailor is being replaced by highly skilled artisans. Oil fuel is labour saving and the amount of physical work performed per man on the average is less and less. So much work of a skilled nature is now required that less men are available for boat work, and men going ashore and afloat go in steam drifters, with a minimum of exertion. The modern man-of-war may, therefore, be likened to a floating factory, full of men doing skilled work, more or less of a sedentary nature, and eating and sleeping in the factory.

Ventilation and heating have vastly improved so that air conditions are infinitely better than in former years.

The factors influencing metabolism in the three periods may be summarized :—

(1) Food, rough and monotonous, split peas and raisins in lieu of fresh vegetables. Abundance of manual labour in the open air. Cooking arrangements primitive.

(2) Food, less rough and less monotonous. More fresh food. Improved cooking arrangements and less manual labour in the open air.

(3) Diet generous and varied, from general messing system and preserved food from canteens. Still less roughage in food and difficulty in obtaining fresh vegetables abroad. Also fruit and vegetables are expensive at home and men have to be fed at a contract price.

Cooking arrangements are much more elaborate and the modern sailor's diet ashore is comparable with the diet of the skilled artisan town worker. In former days, the diet resembled the old institutional diets in its monotony and cheapness.

Finally, there is the increase of sedentary work on board and with it the change in dress. In the old days, the vast majority of men in the Navy were dressed as sailors, with free ventilation at the neck, the only tight clothing being round the waist. This is an ideal costume, more particularly for men living in spaces which it is difficult to ventilate efficiently. In those days the chief site of boils was round the waist under the old cholera belt. To-day an increasing percentage (roughly 30 per cent.) of men wear collars, with the result that boils frequently appear at the back of the neck. In the old days the man in the tropics had the same amount

of fats and protein as the man in the North Sea, whereas now it is possible to vary the requirements for the various stations.

Graph I (fig. 1) shows the average rate for three years in the first period and the present time for diseases of the skin, and diseases of the digestive system on the various stations. It will be seen that in both periods the curves roughly are similar, and that the highest rates are on the China and East Indies stations, with heat, humidity, and the difficulty in obtaining fresh vegetables and fruit.

The Senior Medical Factory Inspector at the Home Office informed me recently that no undue incidence of skin troubles had been noted in the hot and humid cotton-weaving sheds in this country. This fact suggests that climatic conditions are not the primary cause of the common skin infections.

Graph II (fig. 2) shows the same curves for 1921-23 on the various stations, and

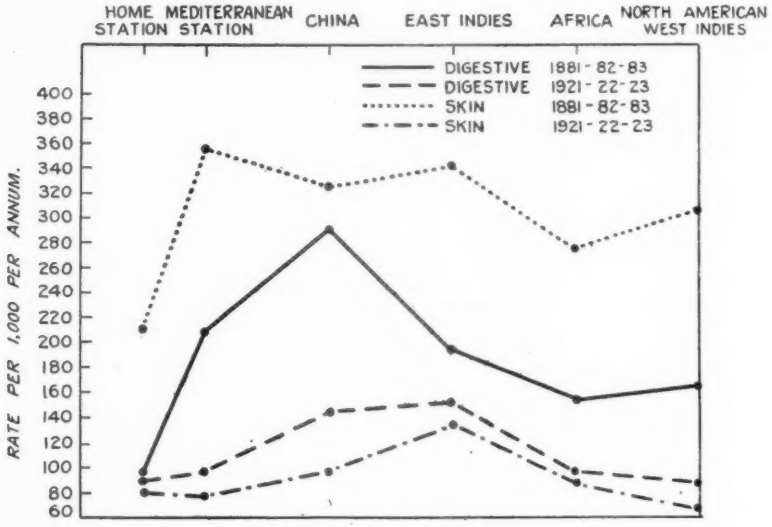


FIG. 1.

also shows the curve for catarrh. In examining nosological returns some years ago I was impressed with the fact that catarrh rates were highest in the summer months and lowest in the coldest months of the year in England. That the incidence of catarrh in the Navy increases with humidity and heat is shown by this graph. The nosological heading "catarrh," is, I believe, peculiar to the Navy and refers mostly to cases of malaise, with no apparent cause, in which the men recover in forty-eight hours with low diet and a purge. Such cases in civil life are sometimes called influenza, and in my opinion are due to increase of intake over output, viz., generous diet and inefficient metabolism. From the above figures and graphs, it would appear that with the elimination of the old monotonous, rough diet of salt meat, ship's biscuit, and lack of fresh food, we have, to a very great extent, lost the diseases peculiar to the Navy in the last century, and now with the introduction of fresh foods, and less

roughage, our diseases are coming into line with those of the civil population, the urban rather than the rural. This is shown in the increased rates for appendicitis and peptic ulcers in the following table.

TABLE II.—Rate per 1,000 per annum for the Total Force.

Year	Appendicitis	Gastric ulcer	Duodenal ulcer
1914	2·0	0·20	0·10
1915	2·1	0·25	0·13
1921	3·5	0·26	0·21
1922	3·6	0·22	0·26
1923	4·2	0·57	0·35

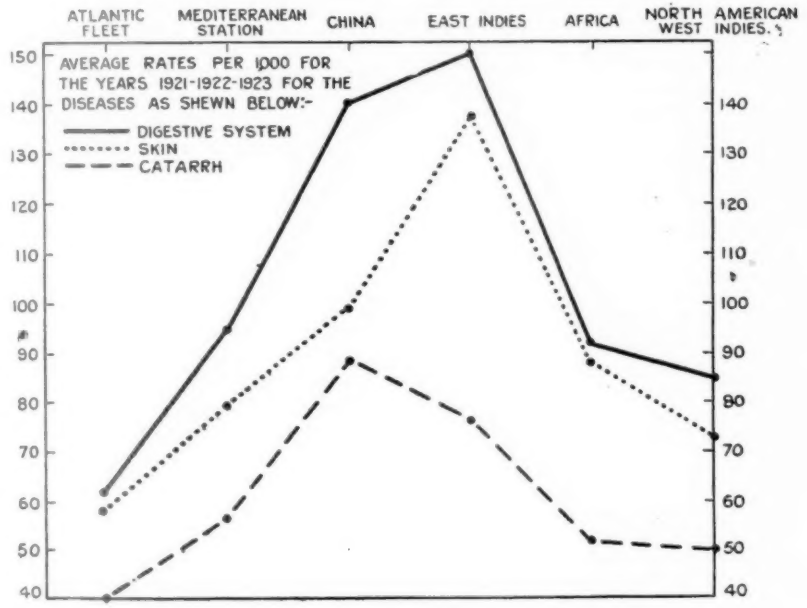


FIG. 2.

These figures are supported by the following figures obtained from the operating room register at Haslar Hospital :

TABLE III.—Figures obtained from the Operation Register at R.N. Hospital, Haslar.

Period of 33 months	Appendix	Gastric and duodenal perforations
1910—1912	171	5 gastric, duodenal nil
1923—1925	320	31 gastric, 5 duodenal

In the first period the average age for appendicitis was 24 years, and in the second 27 years.

In the gastric perforations, the average age has decreased from 32 to 29 years, but with the few cases this is not of much value. The figures show, however, that peptic

ulcers are increasing more rapidly than appendicitis, and also that the rate of perforating ulcers is increasing more rapidly than the rate for ulcers as a whole.

The question at once arises, are the diseases increasing more rapidly in the British Navy than in other communities? Taking the England and Wales male civil population for 1923, age 15 to 45, the mortality rates per 1,000 are: gastric 0·057, duodenal 0·032; and in the Navy the incidence rates for 1923 are: gastric 0·57, duodenal 0·35; suggesting that the relative rates of gastric to duodenal ulcers are much the same in both the civil and naval populations.

The Annual Report of the Surgeon-General of the United States Navy gives the following figures for 1923:

			FRESH CASES.				
Ulcer of stomach ...	18	...	Rate per 1,000 ...	0·15	...	Operations ...	3
,, duodenum...	87	...	,, ,, ...	0·31	...	,, ...	8

These figures are interesting in that the incidence of duodenal ulcers is double that of gastric, whereas in the British Navy the case ratio, and, in the British civil male population, the mortality rates are the exact opposite. The incidence of duodenal ulcer in England and Wales is higher than that of gastric ulcer in males of all ages. Another interesting point is, that in 1924, in the U.S. Navy, there were 748 appendices removed, which gives a rate of 6·4 per 1,000. In the American Navy it would appear that the appendix rate is much higher than in our Navy. The peptic ulcer rate is lower and the rate for perforated peptic ulcers is also lower.

I do not know the American sailor's diet, but from the distribution of their fleets it would appear that a much greater proportion of American sailors are in temperate climates than is the case in the British Navy, and, I believe, the American sailor is better paid than ours. These factors suggest more fresh food and better metabolism.

The Registrar-General's mortality-rates as a whole are not of much value in comparing the incidence of diseases, but when the mortality rates are taken at age groups some use may be derived from them. In the past eleven years the civil appendix mortality-rate has only increased from seventy-eight to eighty-three per million males. But from the graph herewith shown (fig. 3), the rate has decreased below the age of 30, and increased above 30. This I believe shows that in the male civil population appendicitis is increasing in middle and old age.

Similarly, in the next graph (fig. 4), showing the mortality-rates from gastric ulcer, males 1906-1910, males 1921-1923, and females 1921-1923, we see that the death-rate is increasing rapidly in males from 30 to 65 years of age, and that the death-rate is double that of the female.

On the supposition that a rural life with open-air work, and simple food, is healthier than a town life, I have worked out the death-rates for male populations in the age-groups 15-25, 25-35, 35-45 years from Registrar-General's returns for the year 1923.

TABLE IV.

	Gastric ulcer			Duodenal ulcer			Appendicitis		
	15-25	25-35	35-45	15-25	25-35	35-45	15-25	25-35	35-45
England and Wales ...	1·6	4·6	12·3	0·97	3·0	6·4	9·6	6·7	6·5
London	1·9	4·19	16·0	0·27	3·5	6·6	9·9	4·5	5·5
City boroughs	2·03	4·09	13·9	0·83	2·9	7·5	9·1	7·6	5·1
Urban districts ...	1·4	5·1	12·5	1·2	3·4	7·6	11·6	6·8	9·1
Rural districts ...	1·7	4·02	9·8	1·01	1·9	3·7	8·5	6·7	7·1

These figures show that the death-rate from appendicitis and peptic ulcers is lowest in rural districts, and that these rates become relatively lower with increasing age.

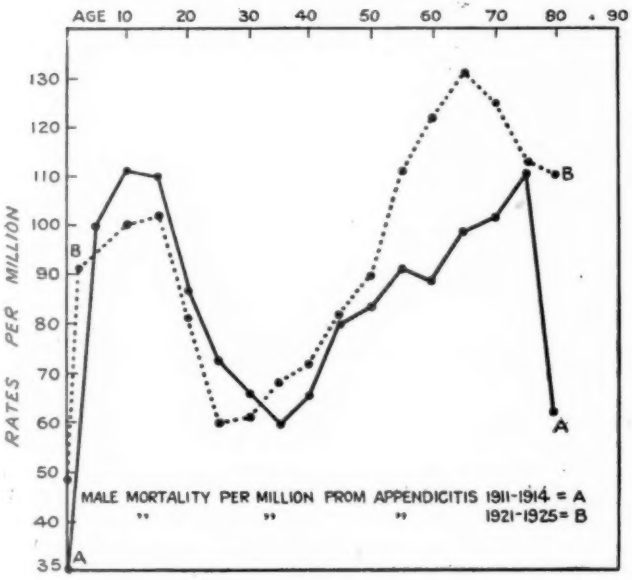

AGE 10 20 30 40 50 60 70 80 90
RATES PER MILLION
130 120 110 100 90 80 70 60 50 40 35
B
A
B
A
MALE MORTALITY PER MILLION FROM APPENDICITIS 1911-1914 = A
" " " 1921-1925 = B

FIG. 3.

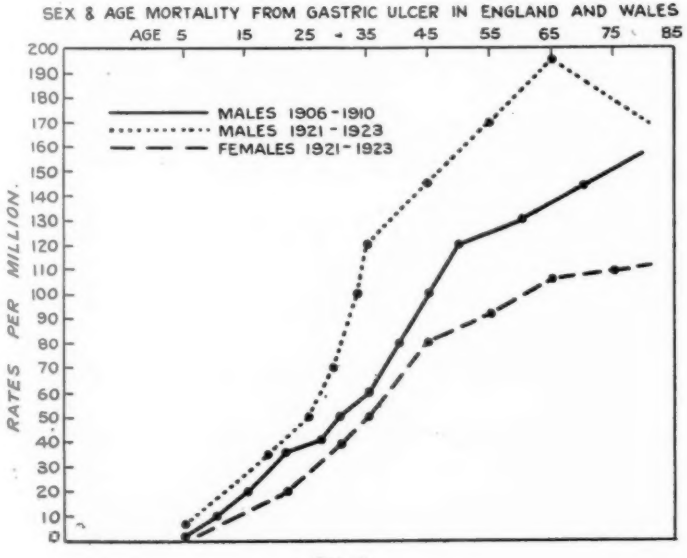

SEX & AGE MORTALITY FROM GASTRIC ULCER IN ENGLAND AND WALES
AGE 5 15 25 35 45 55 65 75 85
RATES PER MILLION.
200 190 180 170 160 150 140 130 120 110 100 90 80 70 60 50 40 30 20 10 0
MALES 1906-1910
MALES 1921-1923
FEMALES 1921-1923

FIG. 4.

44 Dawe: *Diet and Disease in the Navy*

It is, I believe, generally accepted that the predisposing and exciting causes of appendicitis are to be found in our modern diet, with its lack of roughage.

The causes of peptic ulcer, however, are still under discussion. As their incidence presumably varies with different occupations, I inquired of the Registrar-General for the occupational mortality-rates, and was informed that such rates are being worked out for the ulcers and appendicitis, but are not yet available. Assuming that the exciting causes are microbic emboli plus gastric acidity, it appears to me that the predisposing causes connected with the nutrition of the mucous membrane of the digestive tract are also of the utmost importance.

The male gastric mortality-rate being double that of the female may be explained to some extent by the modern dress with free ventilation, as suggested in the case of chlorosis.

In the thirty-five cases of perforated ulcer operated upon at Haslar, the incidence amongst men dressed as sailors is roughly half that of men dressed in collars and coats. Also it would appear reasonable to suggest that alcohol, hot sauces and tobacco exercise an influence. The naval man of forty years ago was much more alcoholic than he is to-day, and practically every man chewed tobacco. Therefore the exciting causes appear to have increased in recent years. The source of microbic emboli is said to be some focus of low grade suppuration, as the appendix, gall-bladder, or teeth. The civil male mortality graphs show the rapid rise in later life of both appendix and gastric ulcer rates, both rising to the age of 65 years and then falling, and we know how frequently gall-bladder inflammation and appendicitis are associated with peptic ulcers. The suggestion that the apices of devitalized dental root-canals may provide a focus can to some extent be examined in the Services from records of dental treatment. At the beginning of the century naval medical officers were responsible for dental treatment. Then we had a few civilian dental surgeons attached to depôts and training establishments, who formed the nucleus of the naval dental service. During the Great War 84 temporary dental surgeons were employed who, after the war, were gradually demobilized—leaving 24 permanent dental surgeons in 1921. In 1922 there were 31 dental surgeons; in 1923, 43; and at the present time, 48. With a decreasing personnel, the naval dental service has increased, so that now we have one dental surgeon to every 2,000 men. In the American Navy in 1924 with 154 dental surgeons, the proportion is one to every 750 men, and this rate is considered inadequate.

TABLE V.

	R.N. 1922	R.N. 1923	U.S.N. 1913
Dental surgeons	31 ...	43 ...	154
Complement	96,560 ...	89,100 ...	116,000
Permanent fillings	27,269 ...	44,089 ...	75,877
Extractions	17,004 ...	19,414 ...	23,341
Pulp treatment	3,639 ...	6,535 ...	8,474
Root treatment	4,974 ...	7,068 ...	6,892

The figures available are shown in Table V, and various interpretations may be put upon them.

With the increase in dental staff in the Royal Navy in 1923 it will be seen that root treatments have increased nearly 50 per cent. in 1922, with relatively fewer extractions. This suggests that a few years ago, with fewer dental surgeons and a much larger complement in the Navy, much less root treatment was possible. In the American Navy, with nearly three times the number of dental surgeons to the same number of men, the root treatments are fewer than in our Navy but a greater

proportion of men receive permanent fillings. This suggests that in the American Navy, with an increased complement of dental surgeons, more teeth can be dealt with in the early stages of decay.

Whatever the causes of peptic ulcer may be, I would submit that the predisposing causes are at least as important as the exciting. For in women with a relatively low rate, we have much the same rates as men for gall-bladder and appendix troubles. Possibly women, as a whole, take more care of their teeth and do not have so much root treatment. The predisposing factor must be greater in men, and from the increase in peptic ulcers in the Navy it would appear that these factors are more marked than in the civil rural population.

If we could discover whether these factors were due to diet, e.g., lack of roughage, to modern cooking, to dental troubles or to inefficient metabolism from men working below decks in ships, something might be done to check the increasing incidence of these acute abdominal troubles. They are serious enough on land with a hospital available but infinitely more difficult to contend with in a ship at sea.

As the general working conditions in the Army and Air Force are very different from those of the Navy, their figures will, I hope, throw more light on the causes of these acute affections of the gastro-intestinal tract. For it is only in the Services that we can get incidence rates and know exactly the conditions under which men live.

Discussion.—Surgeon Captain SHAW, R.N., said: Commander Dawe has shown us that whilst the incidence of diseases of the digestive system generally in the Navy has fallen by approximately 100 per cent. during the last forty years, the incidence of three very important diseases of this system, viz., appendicitis and ulcer of the stomach and duodenum, has increased. A most important consideration is: What is the cause of the increase, because diseases of the intestine, largely appendicitis, are now one of the four chief causes of deaths in the Navy?

I am inclined to believe, as Commander Dawe suggests, that the change in the sailor's life from one which included plenty of open air and a considerable amount of exercise, to the present-day conditions under which there are fewer opportunities for exercise and more time is spent between decks, has something to do with the increased incidence of these diseases.

It may be of interest to note, in this connexion, that during the past thirty years, when the incidence of practically all classes of disease in the Navy has fallen markedly, the incidence of malignant disease, which in man affects chiefly the digestive system, has increased. During the years 1900-1909 the average yearly incidence for malignant disease was 0·10 per 1,000; 1910-1914, 0·2; 1915, 1921, 1922 and 1923, 0·21. The highest incidence was in 1915 (0·37). This can be explained, I think, by the increase in the number of older men serving in the Navy following mobilization. As regards the reduction in skin diseases, boils, etc., I think an important factor has been at work here which Commander Dawe omitted to mention when describing the changed conditions of the sailor's environment. During the period he reviews washing facilities have very materially increased, and the cleanliness of the person and clothes which can now be ensured must help very materially in keeping the incidence of skin affections low.

Commander Dawe has referred to the general messing system now in use in some of our ships and establishments. The introduction of this system (first tried out in our first *Dreadnought*) is an important landmark in naval hygiene and represents the latest of the many changes which have been introduced to improve the sailor's diet. It should be noted (Commander Dawe does not make this point sufficiently clear, I think) that in the general mess system the amount of money available per head is the value of the standard ration *plus* the messing allowance. This amount is at present 1s. 5d., and for boys 1s. 6½d., per day.

Major G. D. JAMESON, R.A.M.C., said that Surgeon Commander Dawe had expressed a hope that the experiences in the Army and Air Force would throw some light on the problem. As far as the Army was concerned, the health figures gave little help except that in the main they agreed with the Navy figures. As suggested by Commander Dawe, life in the Army had

46 Dawe: *Diet and Disease in the Navy*

not altered so appreciably during the last forty years as it had in the Navy, except for the fact that the soldier was better housed and clothed. As regarded diet, the improvements had been mainly in the direction of a better-balanced and more varied diet than in actual quantity, the caloric value of the diet forty years ago being about 3,000 as compared with the present-day value of 3,110. It was interesting to note the high caloric value of the Navy forty years ago as compared with the Army, and also the present United States Army ration of over 5,000 calories as compared with the British. As regarded actual sickness rates, it was pointed out that although the Army rates per 1,000 were somewhat lower generally than the Navy, the same relative decrease in digestive diseases had taken place in the last forty years, and also that since 1910 there had been a rise in the attack rate of appendicitis and peptic ulcer. As regarded the dental point of view, it was apparently the opinion of Army dental officers that a definite connexion between a man's dental condition and his tendency to peptic ulcer was hard to trace, the incidence of this disease not being appreciably different in young soldiers who had been under a dental officer's care since enlistment and old soldiers who had not had the advantage of skilled dental treatment during the whole of their service. He (the speaker) inclined to the belief that the rise in appendicitis and peptic ulcer must, therefore, be looked for in the present-day diet with its large percentage of chemically prepared and purified foods.

Fleet Surgeon W. E. HOME, R.N., said that perhaps the author ascribed too exclusively the improvement in health he had brought out to the recent improvement in the feeding and conditions of the sailor since 1912. He (Fleet Surgeon Home) had served in the *Galatea* in the Home Fleet in 1901-2, and the average of sickness by the Sunday signals was, "men sick and in hospital, 4 per cent." When he was Senior Medical Officer in the Channel Fleet, in the *Exmouth*, 1905-7, the similar average had fallen to 2, the rate thus being halved. At the time he had thought the improvement was due to the larger cubic space and better ventilation of the newer pattern of ships. He was quite sure that while he was a student and during his early service there were very few cases of appendicitis; it was not that they were merely unrecognized. It was very important in reporting statistics of illnesses and deaths in the Service to state the ages of the patients concerned, and also of the men in the Service generally, which seemed, by recent reports, to be older now than in former years. He directed attention to a paper in the February number of the *Military Surgeon*, by an officer of the U.S. Public Health Service (p. 237), stating that Japanese research workers could produce experimental gastric ulcers by means of diet, and suggesting that tuberculosis depended in some degree on diet, and that the incidence of pulmonary tuberculosis in a community varied inversely with the consumption of milk by the inhabitants.

Lieut.-Col. P. H. HENDERSON, R.A.M.C., said that Surgeon Commander Dawe had done well in drawing attention to certain disabilities which, owing to the increase in their mortality apart from the noticeable increase in their incidence, were attracting much attention in the Services.

Perhaps there had been greater changes in recent years in the environment of the sailor, but there had also been very considerable changes in that of the soldier. Commander Dawe appeared to think that there was perhaps some difference between the environment of the sailor and the soldier which accounted for the increasing incidence and mortality in the Navy from appendicitis, gastric and duodenal ulcers. But he (the speaker) could assure him that while there was a diminution in the incidence of diseases of the digestive system as a whole in the Army, as there was in the Navy in the periods under discussion, there was an increase at any rate in the incidence of appendicitis, gastric and duodenal ulcers in the Army corresponding to that in the Navy. This increased incidence was shared also by civilians and was attributed to the more accurate diagnosis of the present day. This explanation could not be accepted, for whereas life in the Navy had become less strenuous, it was not so in the Army or in civil life. Factors common to all three communities must be sought as an explanation of this increased incidence. Two possible factors were: (1) The artificial nature of modern food—the adulteration of bread was a signal instance; (2) the extraordinary increase in cigarette smoking. He did not overlook the fact which Surgeon Commander Dawe pointed out, namely, that in the Navy tobacco chewing was formerly almost a universal habit, but in his opinion chewers did not swallow the tobacco juice; they spat it out, whereas cigarette smokers freely inhaled the smoke and with it the nicotine.

This had an important influence in disorganizing the sympathetic and vagus mechanism so intimately concerned in digestion.

In regard to boils and other septic skin conditions, he (the speaker) used to search for a possible cause in the shortage of vitamins owing to lack of green vegetables or faulty cooking, but he had been unable to trace any definite connexion between the two. In the Army these conditions were most prevalent in humid, hot stations with little air movement, like West Africa and Jamaica, where the troops were frequently bitten by mosquitoes, and in one or two home stations in summer, where the men got hot and dirty grooming horses, etc. He agreed with Surgeon Captain Shaw that the actual cause of these boils and skin complaints was the want of hot water and soap for thoroughly cleansing the skin under these hot, humid conditions.

Surgeon Commander DAWE, in reply to Group Captain Cooper, said that owing to the benefits derived from school dental service recruits joining the Navy were dentally more fit than they used to be. In reply to Surgeon Captain Shaw, who associated the great decrease in skin troubles in the Navy with the much improved personal washing arrangements in recent years, he said he considered that this was not the primary factor, as young officers in the Navy, who could not be accused of lack of cleanliness, not uncommonly developed boils at the back of the neck. He was pleased to hear that the Navy was no worse off than the Army in the incidence of appendicitis and peptic ulcers. The increase in the Navy in appendicitis and gastric and duodenal ulcers was a matter of the utmost concern to naval medical officers, for it followed that in a battleship with a complement of 1,000 or more, in the course of a year's cruise there was the probability of four cases of "acute abdomen" occurring from appendicitis or peptic ulcers. In this connexion he was glad to hear the question of cigarette smoking raised, as he firmly believed it was an important factor in the incidence of peptic ulcers.

JOINT DISCUSSION No. 7.

Section for the Study of Disease in Children and Section of Medicine.

Chairman—Dr. J. D. ROLLESTON (President of the Section for the Study of Disease in Children).

DISCUSSION ON THE SERUM TREATMENT OF SCARLET FEVER.

Dr. E. W. GOODALL.

IN initiating a discussion on the serum treatment of scarlet fever, there are two points which I wish to mention before coming to the subject-matter proper to the discussion; the first is that I cannot claim to deal with the question in an authoritative manner, because before I had thoroughly made up my mind as to the efficacy of the treatment I ceased to enjoy first-hand opportunities for so doing. The second, and more important, is that I shall assume that the hæmolytic streptococcus of George and Gladys Dick is the true bacterial cause of the disease, for I am of the opinion that the Dicks' claim has been established. If there be any clinician who, reporting favourable results from the serum, yet denies that the above-mentioned streptococcus is the cause of scarlet fever, he must explain his success by some other reason than that advanced by those who believe in the specificity of the micro-organism.

Now clinicians, in endeavouring to ascertain by personal observation, and by statistical results, the value of the serum treatment of scarlet fever, are not so favourably placed, at any rate so far as those practising in this country are concerned, as were those who were called upon to pronounce an opinion on the antitoxin treatment of diphtheria upwards of thirty-two years ago. At that time, in London, the type of that disease was very severe, as the fatality of the cases admitted to hospital was over 30 per cent., so that it was comparatively easy in a few weeks to come to a decided conclusion on the value of any sort of treatment. But with scarlet fever the case is different, because for some years past the type of the disease has been very mild; consequently a larger number of cases, and a more lengthy period are necessary in order to pronounce a just judgment. The fatality of the cases treated in the Asylums Board Hospitals has only once been above 2 per cent. since 1910, and that was in 1916; at the North-Western Hospital, of which I had charge for the last ten years of my service, the fatality was 1·01 per cent. for the year 1925. There is not much room, therefore, for improvement in treatment so far as the saving of life is concerned, though, of course, the superiority of one method of treatment over another must, and may, be demonstrated in a non-fatal, or slightly fatal, disease by other results than a fall in the case mortality. There is another point which makes it more difficult to arrive at the truth in the case of scarlet fever than in that of diphtheria, namely, that whereas diphtheria in the great majority of cases is a toxæmia pure and simple, in scarlet fever it is admitted that there is a bacteriæmia as well as a toxæmia, and it is often a bacteriæmia of the same micro-organism as produces the toxæmia. In diphtheria, if there is an additional bacteriæmia, it is rarely, if ever, of the *Corynebacterium diphtheriæ*. Further, I understand that those bacteriologists who are responsible for the making of the scarlatinal antiserum do not claim that the serum is antibacterial as well as antitoxic. In a double pathology such as occurs in those very cases of scarlet fever which are to be blamed for most of the deaths—the anginous or septic cases—it is not easy always to decide whether the symptoms are due entirely to septicæmia, or whether toxæmia is also present.

152 Goodall: *Discussion on the Serum Treatment of Scarlet Fever*

It appears to me that the following are the points which can most usefully be considered in this discussion:—

(1) Is the treatment by the new serum better than any other treatment?

(2) If it is, how is its superiority shown?

(3) Can any difference be made out, in respect of results, between the toxæmic and the septic symptoms?

(4) What is the correct dosage?

As already said, it is difficult in this country, at any rate in London, to demonstrate any improvement in the case mortality because of the natural mildness of the disease. So far as I have been able to ascertain from published accounts, only one serious claim has been put forward that there has been any such reduction, viz., that by Cushing, of Montreal. Several observers have stated their belief in the efficacy of the serum, but on other grounds. Even in some cities of the United States there has been a comparatively high fatality during recent years. No statement has been made in published reports on the treatment in respect of the fatality rate, so that it is to be presumed that no reduction was observed; for example, 160 cases, stated to be severe, treated by Woody, of Philadelphia; 35 by Selinger, of Washington, D.C.; an unstated number by Birkhaug, of New York. There have been some high fatality rates amongst hospital cases in New York. Thus, at the Willard Parker Hospital the rates per cent. were as follows for the five years 1919-1923—5·9, 12·0, 8·4, 8·2, and 4·6. Serum treatment was begun as a routine measure in March, 1925, but I have not seen any statement as to the effect, if any, on the fatality rate. Scarlet fever has been extremely severe recently in Eastern Europe, and in China. In the former country the specific serum has apparently not yet been used, at any rate extensively; but in the latter it was tried at the "Government Isolation Hospital," at Peking, and found to be of little value in the toxic form of the disease. Later, a serum produced by a different method proved to be of some use. Cushing, of Montreal, states that at the Alexandra Hospital, Montreal, the scarlet fever fatality was over 5 per cent., but that it had been falling, and that in 1923 it was 3·2 per cent. In 1924, when serious cases were treated with convalescent serum, the fatality was 2·0 per cent. Writing in August, 1926, he stated that since antitoxic serum had been more generally used, 500 out of 800 cases had been treated with it and that there had been ten deaths, a fatality of 1·2 per cent. In eight of these cases, however, death was not due to scarlet fever, and in the other two the serum was not given until the ninth day. With the exception of this series of cases, I have not been able to find any evidence of a reduction of the fatality, such as was the case in diphtheria immediately after the introduction of the serum treatment, and those clinicians who have come to the conclusion that the treatment is valuable have done so on other grounds. Amongst these must be mentioned Harries, of Birmingham, Thomson, of London, and Hutchinson, of Dublin, besides the American observers referred to above, to whom must be added Park, of New York and Gordon, of Chicago. The principal grounds on which they base their opinion are rapid fall of temperature, disappearance of delirium, rapid subsidence of the faucial inflammation, quicker fading of the rash than is usual, and lessening of the amount of desquamation: briefly, a more rapid improvement in the condition of the patient than is met with in other methods of treatment. It has been noticed, however, by more than one physician, that now and again there is a case that does not respond. All agree that, as with diphtheria antitoxin, the earlier the remedy is given the better and the surer the result, and that if it is administered late, on the fifth day or after, favourable effects are hardly to be expected.

My own personal experience has been very limited. At the North Western Hospital we began to treat a few selected cases towards the close of 1925. I found it very difficult in that desultory way to come to any definite conclusion; moreover,

the cases which were being admitted at that time were very mild. I remember having a series of 300 admissions without a death. About April, 1926, we began to treat rather more cases of a more severe type, and were obliged to make some selection, for we were not supplied with an unlimited amount of serum. Up to the time I left the hospital in August, I had personally observed and made notes upon fifty-six cases. This is far too small a number upon which to make a final pronouncement, and I can give only a preliminary impression. Patients in the more severe cases received serum; a few cases were mild on admission, but became worse in the course of a day or two and then were treated.

Of the fifty-six patients four died, but not one of them from scarlet fever, pure and simple, and uncomplicated.

The first was a boy, aged 1 year, who had severe faucial inflammation and bronchitis. 25 c.c. were given on the third day and 20 c.c. on the fourth. There was no improvement at all and he died on the fifth day. Bronchitis was chiefly responsible for his death.

The second case was that of a boy, aged 5 years, with toxic symptoms. He received 20 c.c. on the fourth day and 25 c.c. on the fifth. Two days later there was an improvement; but septic complications, dacryocystitis and otitis media developed, and he died on the fifty-second day from cerebral abscess.

The third fatal case was that of a boy, aged 7. He received 50 c.c. on the third day, but showed no improvement. Otitis, mastoiditis and endocarditis supervened, and he died on the twenty-sixth day.

The fourth case was that of a woman, aged 28, who had been confined four days before admission and two before the rash appeared. She exhibited very severe toxic and septic symptoms; the latter were due to uterine infection, as there was no exudate on the fauces. She was given 45 c.c. on the second day and 45 c.c. on the third. Her condition improved on the fourth day so far as the toxic symptoms were concerned. She died on the thirteenth day. A post-mortem examination was refused, but I have no doubt that she had pyæmia, of which the focus was in the uterus.

Twenty of the patients were under 5 years of age and and thirty-eight were under 10. Forty-one patients received serum during the first three days of their illness.

I have not witnessed any of the remarkable cases described by one or two observers, toxic cases in which all the symptoms abated within twenty-four hours; but amongst my cases there was none that was purely toxic and uncomplicated. If the four fatal cases are excluded, the fifty-two cases comprised two with combined toxic and septic symptoms, thirty-three septic and seventeen benign cases. By septic I mean those cases in which the faucial lesion is a prominent feature of the attack, scarlatina anginosa. I do not mean that all the septic cases were necessarily severe, but those which were not might readily have become so.

Two cases are described as being both toxic and septic:

(1) A girl aged four years and (2) a man aged 34. On the third day of the disease 60 c.c. were given in the first case and 85 in the second. Both patients were much better the next day. The girl recovered without any complication, the man suffered from slight rheumatism.

The following table is compiled from notes made on the progress of the cases which I personally observed:—

Exclusive of fatal cases	Day of disease on which serum was first given: number of cases					
	1	2	3	4	5 and over	Total
Much better next day	4	12	5	2	1	24
Better next day	3	3	3	2	—	11
Better two days after	—	2	5	—	1	8
No improvement	—	—	1	3	5	9
Total	7	17	14	7	7	52
Complications	—	2	6	4	2	14

154 Goodall: *Discussion on the Serum Treatment of Scarlet Fever*

By "no improvement" is meant no improvement that could be ascribed to—and would have been expected from—the use of the serum; the patients gradually improved in the same manner as under the ordinary forms of treatment.

The occurrence of complications was as follows (the four fatal cases are excluded): First-day cases, *nil*; second day, two, one of rheumatism and cervical adenitis, and one of otitis media; third day six, two of nephritis, three of otitis, and one of rheumatism, an old heart case; fourth day, four, one of otitis and cervical abscess, one of nephritis, one of rheumatism, and one of broncho-pneumonia; fifth day and over, two, one of otitis and one of albuminuria (? nephritis).

From the results recorded in this table I came to the conclusion that a *prima facie* case had been made out in favour of the serum. The results, so far as they go, support the opinions expressed by other clinicians that the earlier the treatment is begun, the better the result and the smaller the likelihood of complications.

I noticed that in some of the cases with marked faucial inflammation, while the general symptoms improved quickly, the throat lesion was slower in clearing up.

Twenty-four of the patients underwent an attack of serum-sickness, and in one case there was an abscess at the site of injection. In only one case was the attack severe. The serum was, I understood, an unconcentrated serum, that is, it had not been deprived of its euglobulin and albumin.

It is to be remembered that successful results have been claimed for multivalent antistreptococcal serum in scarlet fever long before the new serum was introduced. I have seen what I believed to be the beneficial results of a multivalent serum. Whether in those cases the good results were due to the accidental presence of the special hæmolytic streptococcus in the mixture of bacteria which was used as the antigen for producing the antiserum, or whether they were due to what is known as protein therapy, is uncertain; probably the former, for in my successful cases I never saw one in which there was a rigor or any shock. On the other hand, I have met with some remarkably favourable results of the treatment of typhoid and paratyphoid fever with both an antiserum and a vaccine. Such results could not have been due to any specific action. In several of the cases a rigor or shock symptoms occurred. In the present series of cases protein therapy may, in my opinion, be excluded.

There is not sufficient evidence to draw any conclusion as to the question of an antibacterial action of the serum. Experiments on animals go to show that the serum supplied up to the present time has no antibacterial value.

As regards dosage, we were told frankly by Dr. O'Brien that the dosage was uncertain, because there was no certain method of standardization. We gave the serum in doses of 20 to 100 c.c., mostly in single doses (forty-five cases). The most frequent dose was 50 c.c. From my experience of these cases, which I admit to be very limited in number, I should recommend that 30 c.c. should be given in cases, if not severe, seen on the first day, but 50 c.c. in severe cases. Twenty c.c. should be added to the dose for every additional day, and according to the severity of the attack. Thus, a severe case seen on the second day would require 70 c.c., and on the third up to 90 c.c. It is advisable to give the dose in one injection only. In nearly all my cases the injections were given intramuscularly; none was given intravenously, but this method should be used in the severe toxic cases where possible. The serum is antitoxic only, and not antibacterial. As scarlet fever is not infrequently also a septicæmia, the cases in which the symptoms are chiefly due to septicæmia will not, presumably, receive benefit.

I do not advise giving the serum in mild cases, as the serum-sickness which may follow may be more unpleasant than the attack of scarlet fever. But every case should be carefully watched, and if it shows signs of becoming worse should then be treated with serum.

So far as I am aware, the serum has not been given in this country by unit-dosage, as in the United States. A unit has been defined as that amount of serum which will neutralize 100 skin-test doses of toxin, and a skin-test dose as 0·1 c.c. of a 1 in 1,000 dilution of the toxin. But as the methods of standardizing the toxin and the serum have so far proved very unsatisfactory, the unit system of dosage has not been adopted in this country. In the United States the dosage has been from 2,000 to 10,000 units. In volume these doses would be from 5 or 6 to 50 or 60 c.c.

I hope that those who follow me in this discussion, and who have enjoyed a larger experience than mine, will deal chiefly with the following questions: (1) Has their experience enabled them to form a decided opinion as to the value of the treatment? (2) If their opinion is favourable, what is the effect of the serum, if any, in respect of the septic element in the disease and of its complications? and (3) What is the dosage and what are the conditions by which it is influenced?

Dr. R. A. O'Brien.

As my own clinical opportunities are limited, I recently referred the three important questions before us to the superintendents of three hospitals—all outside London—because I anticipated that experienced London clinicians would be present at this meeting and also because the disease in the north has probably been of a more severe type than the very mild type commonly seen in London. The three superintendents generously gave me permission to quote their opinions and experience this evening.

In the *Edinburgh Medical Journal*, December, 1926, Dr. Benson, of the City Hospital, Edinburgh, described his results in the treatment of 100 cases of scarlet fever. He concludes that: "The administration of antitoxic serum within the first forty-eight hours of the disease has a very favourable influence on the specific toxæmia of scarlet fever. In relieving the more urgent symptoms of the acute stage it undoubtedly renders the patient more comfortable.

"There are indications that the liability to subsequent complications is diminished.

"The administration of serum even on the first day of illness apparently does not act as an absolute safeguard against the subsequent development of complications of septic type in convalescence.

"In toxic cases of scarlet fever, serum should be administered either intravenously or intramuscularly at the earliest opportunity and the dose repeated if necessary."

Dr. Benson has since treated between two and three hundred patients with English concentrated serum. The dose was usually 10 c.c.—exceptionally 20 c.c. He says: "The former dose exercises a definitely favourable effect in the mild and moderate cases. The serum does not seem to touch the septic type of the disease. Serum rashes were troublesome. Serum therapy is certainly worth while, more particularly in the sharper cases. I am not, however, prepared to discharge my patients in the third week of convalescence merely on the strength of serum treatment."

Dr. Harries, of the City Hospital, Birmingham, writes: "We have used scarlet fever antitoxin for about eighteen months, and concentrated antitoxin as a routine in all except the mildest cases for six to eight months. Serum sickness is infrequent and trifling when it occurs. A dose of 10 c.c. is sufficient in the average case.

"There is some reason for hoping that serum will diminish complications and will reduce the necessary stay in hospital. If it has these effects, it is obvious that the money spent on antitoxin would be well spent.

"It is unnecessary to reiterate the well-known effects of scarlet fever antitoxin on the toxæmia of scarlet fever. The more striking the case the more striking are

the results of treatment with scarlet fever antitoxin. The problem of scarlet fever is, in my opinion, one of late morbidity, and it is impossible to forecast what complications a patient with a mild initial attack may develop in the second or third week. Only a long series of cases treated and untreated with serum can establish whether serum treatment does decrease the incidence of late complications.

"I found no direct curative influence on the septic complications of scarlet fever. A serum on the market, stated to be antibacterial as well as antitoxic, has, in my hands, given no antibacterial results.

"To sum up, I have no personal doubt as to the curative value of scarlet fever antitoxin, and should regard it as a failure of my duty if I withheld antitoxin from a severe case of scarlet fever.

"With regard to prophylaxis, we have never yet known a child who has been rendered Dick-negative by antitoxin to contract scarlet fever, although repeatedly exposed. We have, however, seen children, whose passive immunity had waned and who had become Dick-positive, contract scarlet fever after exposure."

Dr. Harries in a later letter adds that in fifty successive patients treated with concentrated serum there were no septic complications. Thirty-three patients showed no symptoms of serum sickness; amongst those with symptoms none were in any way severe, and all the patients were ready for discharge in thirty days. At present all clinically severe cases receive serum; of the others every alternate patient receives serum.

Dr. Rundle (Liverpool City Hospital) has used serum in about one hundred cases. Five deaths occurred. "The cases were selected for treatment by reason of their 'toxicity,' and many of them would, in our opinion, have succumbed without the serum. The hundred cases includes, however, about twenty-five of the septic variety, coming late under treatment and deriving no obvious benefit from the antitoxin. The deaths, with one exception, occurred amongst this 'septic' type. Antitoxin is of no use once septic throat, adenitis, etc., have set in. The serum phenomena have been more intense than one finds with antidiphtheria serum. Although one expects a dramatic fall of temperature and general alleviation with serum in the toxic cases, there is no guarantee that the usual complications will not arise. We have had some severe suppurative conditions in patients who had serum some days previously."

The experiences of these three observers apparently agree fairly closely with the general experience of clinicians in America.

The question of specificity and standardization of scarlet fever antitoxin arises directly from the important question of dose raised by Dr. Goodall. My colleagues, Dr. Parish and Dr. Okell, have kindly allowed me to show two tables from a paper which they have in preparation. From the first table it will be seen that when a sufficient dose of culture of the hæmolytic streptococcus of scarlet fever is injected intravenously into a rabbit, the rabbit almost invariably dies of the initial "toxæmia" or "septicæmia" in less than two days. Normal horse serum and non-specific antitoxin do not protect the rabbits, human convalescent serum does so to some extent; concentrated scarlet fever serum protects completely when given in a moderate dose. The most interesting part of the table is that relating to the use of the serum that has been issued commercially by many laboratories for many years past as antistreptococcus scarlet fever antiserum. This serum was a legacy from the period about 1902 when Marmorek, Gabritchewsky, Moser, Schick and others used the serum of horses injected with broth culture of streptococci obtained from the throats of scarlet fever patients for the treatment of scarlet fever. There is very little doubt, from their clinical records, that the serum they used at first had a definite therapeutic effect on scarlet fever. The serum later slowly

somewhat declined in favour because some workers failed to record any improvement in their patients after the use of the serum, and further, using large doses of the serum, they found that their patients often had severe serum reactions. The incidence of these serum reactions to-day has been greatly reduced by the well-known processes of concentration. Examination of some of the serum made by this method has shown that it has a definite but low amount of antitoxin in it.

Dr. Parish and Dr. Okell have shown that some of this serum was approximately half as strong as a high value concentrated scarlet fever antitoxin of to-day. Unfortunately, it does not seem to have occurred to anyone to use an obviously simple method of titrating this serum, i.e., to find accurately how much of this serum was necessary to protect from scarlet fever contacts exposed to infection. Had this been done, it is fairly certain that some of the serum in use could easily have been demonstrated to contain a very small amount of antitoxin and would have been replaced by the most potent serum then available.

The second table relates to some very recent work. A fascinating immunological puzzle concerns the relation of the hæmolytic streptococci to one another. The hæmolytic streptococcus of follicular tonsillitis and that of puerperal septicæmia cannot with any certainty be distinguished by the bacteriologist from the streptococci found in every scarlet fever throat. An obvious method of attack on this problem is to make toxins from the various organisms and test them on human beings, to see if the same patients are positive to all three toxins or negative to all three toxins. And, if so, to discover whether the positive Dick reaction is neutralized by anti-follicular tonsillitis antoxin or by anti-puerperal, anti-cellulitis, or other antitoxin.

Such a research has long been projected by Dr. Okell, and, as opportunity offers, is being carried through. But while waiting for the results of tests on human beings, Dr. Parish and Dr. Okell have carried out in rabbits the experiment indicated. It is remarkable that six strains of hæmolytic streptococci from patients suffering from puerperal septicæmia and two strains from patients with follicular tonsillitis, which promptly kill all the unprotected rabbits, fail to kill within three days any of the rabbits protected with scarlet fever concentrated antitoxin. The antigenic overlap amongst these hæmolytic streptococci must be very close, amounting perhaps to identity. It is reasonable to hope from these results that the homologous antitoxins, and also the concentrated scarlet fever antitoxin, may be of definite service in combating at least the first toxæmia or septicæmia of follicular tonsillitis, puerperal septicæmia and cellulitis, etc., caused by this same group of hæmolytic streptococci.

The question of *dosage* is closely linked with that of standardization. The various methods are well known, i.e., neutralization of toxin in the skin of human beings by measured quantities of antitoxin, similarly in goats, the determination of an adequate dose for passive immunity, the dose necessary for Schultz-Charlton blanching, and the rabbit protective dose. The determination of the prophylactic dose, i.e., one that will turn positive Dick reactors negative overnight and keep them negative during the incubation period of scarlet fever, has proved to be easy and supplies a method available in the hands of the hospital superintendent for titrating the serum he proposes to use, should he so desire. It is found that, with a good concentrated serum, this dose varies from 2 c.c. to 4 c.c. or 5 c.c. Such a serum will usually produce blanching when injected into an early scarlet fever rash in a dilution of upwards of $\frac{1}{1000}$.

Conclusions.

Concentrated scarlet fever antitoxin is of use in the treatment of scarlet fever. No antitoxin or other serum at present available has any direct action on septic

158 O'Brien: *Discussion on the Serum Treatment of Scarlet Fever*

complications. The early use of serum probably reduces the liability to late septic complications and reduces the length of stay in hospital, but many further observations are required.

Concentrated scarlet fever antitoxin protects rabbits against the first "toxæmia" or "septicæmia" caused by hæmolytic streptococci obtained from cases of follicular tonsillitis, puerperal fever, cellulitis, etc. The antigens of these various cultures must bear a close relationship to each other.

TABLE I (PARISH AND OKELL).—RABBITS INJECTED INTRAVENOUSLY WITH 10 C.C. OF A TWENTY-HOUR DIGEST BROTH CULTURE OF SCARLET FEVER STREPTOCOCCUS. SERUM INJECTED INTRAVENOUSLY FOUR TO SIX HOURS PRIOR TO INJECTION OF CULTURE.

	Total rabbits	Percentage living six days after injection of culture. 5 c.c. or more	1·0 c.c.	0·25 c.c.	0·1 c.c.
Normal horse serum ... ... } Concentrated diphtheria antitoxin }	20	5	—	—	—
Unconcentrated anti-streptococcus serum erysipelas ...					
" " " strangles ...					
" " " measles ...	29	17	—	—	—
" " " puerperal ...					
" " " pyogenes (Swann)					
" " " " (Shilling)	17	29	0	—	—
" " " S[1] "	6	50	—	—	—
Serum from scarlet fever convalescents (Dick-negative) ...	6	50	—	—	—
Unconcentrated scarlet fever antitoxin (early stage of immunization), batch "P"	9	66	0	—	—
Unconcentrated scarlet fever antitoxin (late stage of immunization), batch "Q"	15	77	33	0	—
Concentrated scarlet fever antitoxin, batch "R"	15	88	66	33	—
" " " " " "S"	12	100	100	66	33

TABLE II (PARISH AND OKELL).—RABBITS INJECTED WITH SERUM INTRAVENOUSLY FOUR TO SIX HOURS BEFORE INJECTION OF CULTURE INTRAVENOUSLY.

Culture, 5 to 20 c.c.	Total number of rabbits	Unprotected: No serum or normal horse serum or concentrated diphtheria serum	Protected: Streptococcus antitoxic serum: Concentrated scarlet fever, 5 c.c.	Concentrated puerperal, 5 c.c.	Unconcentrated pyogenes, 10 to 15 c.c.	Unconcentrated erysipelas, 10-15 c.c.
Tonsillitis	24	0	77	—	33	—
"	9	0	100	—	33	—
Lymphangitis ...	9	0	100	—	66	—
Fatal septicæmia ..	12	0	33	—	66	—
Severe septicæmia ...	15	0	66	—	—	—
Endocarditis ...	12	0	83	—	—	—
Puerperal fever (uterus) ...	25	10	83	16	0 (conc., 5 c.c.)	—
" " ...	25		100	50	33 (conc., 5 c.c.)	—
" (blood) ...	11	0	100	100	—	—
" " ...	9	33	66	66	—	—
" (uterus) ...	11	0	100	33	—	—
" (blood) ...	11	0	100	33	—	—
Erysipelas	36	8	70 (conc., 5 to 15 c.c.)	—	—	77

Dr. F. FOORD CAIGER

said that he, too, had not had the opportunity of trying the serum, as there had not been under his care cases suitable for the purpose. When it was realized that to-day the mortality of scarlet fever was 1 per cent., it would be seen that only in a long series of cases could one find much indication of the effect of the serum in saving life, otherwise the only indication was that afforded by the rapidity with which the

[1] Broth cultures of scarlet fever streptococcus included in antigen for immunization.

symptoms cleared up, and the extent to which complications developed. Dr. Goodall had evidently noticed a rapid clearing up of symptoms in cases not necessarily severe; but with regard to the incidence of complications the results were not convincing. This impression gathered confirmation from the records quoted by Dr. O'Brien of the work of Dr. Harries, Dr. Rundle and Dr. Benson. Therefore in that respect one could not get very far. The success of the attempts made by earlier pathologists had not been greatly exceeded by the success attending the use of the present serum. He (the speaker) remembered a paper recording the work of Palmirski and Zebrowski, who, in (he believed) the late nineties, had tried a multivalent serum in a thousand cases of children in a Warsaw hospital with satisfactory results. They reported that in the mild cases it usually acted like a charm, but in the severe cases, many of which were undoubtedly septic, its good effect was only moderate. They went so far as to say that if streptococci were found in the actual circulation the serum was of no value at all. Their serum was prepared from seventeen fatal cases of scarlet fever. Moser's work, published in about 1900, achieved for the author an almost world-wide reputation; but his serum treatment had gone the way of so many others.

With regard to Dr. Goodall's statement that in certain cases in which septicæmia was present in scarlet fever the hæmolytic streptococcus of Dick was the organism found, he (the speaker) was under the impression that in those cases which had been examined it was, almost invariably, some organism other than the Dick coccus which was present in the circulation.

He believed that success with antitoxic serum to-day would chiefly be obtained in the simple form of the disease, and would consist in a shortening of the attack; and that in the toxic form, which was now rarely seen, some benefit might be anticipated. One would hardly expect that the Dick serum alone would be of much use in the septic variety of scarlet fever. In that class of case it would seem that one had to give a multivalent serum in addition.

He would briefly refer to the success he himself had obtained between twenty and thirty years ago with a serum which was sent to him from Paris by Professor Besredka, of the Institut Pasteur—a multivalent serum derived from twenty strains of scarlatinal streptococci. The Professor had sent him a large case of it, and it almost seemed as if there was enough to last for life, so large was the number of bottles of serum in 50 c.c. doses. It was obvious, however, that he could not use it in every case of scarlet fever, and he decided to give it only to patients whom he regarded as likely to die under ordinary treatment. The dosage employed was 50 c.c., repeated daily for so long as necessary, the average number of doses given being between three and four. Enough had been sent for the treatment of forty-four cases, and the ultimate mortality of those very severe cases—in each of which a fatal prognosis would not have been otherwise far wrong—had been only 36 per cent. The mortality-rate in cases in which the treatment was begun on the fourth and fifth days of the attack was 23 per cent.; when it was given two days later, the rate was 35 per cent.; and when it was not given until the ninth day the death-rate was just over 50 per cent. This was more than twenty years ago, and he (the speaker) doubted whether the treatment of scarlet fever had advanced very much further since that time.

Dr. J. D. ROLLESTON

said that Dr. Goodall had referred to the mild character of scarlet fever in this country, and in that connexion the following figures might be of interest. From 1874 to 1879, shortly after the hospitals of the Metropolitan Asylums Board were established, the case-mortality from scarlet fever was 13·9 per cent., which was

160 Rolleston: *Discussion on the Serum Treatment of Scarlet Fever*

exactly the figure given by Chodzko,[1] the Polish epidemiologist, of the mortality-rate in Warsaw during the first twenty-six weeks of 1926. During the following years the case-mortality in London gradually declined. For instance, in the period 1890-94 it had fallen to 6 per cent., and in 1900 it was 3 per cent. From the last Report of the Asylums Board (1925-26) it would be seen to be 1·1 per cent; while in his (the speaker's) own hospital (Western Hospital) it was 0·65 per cent. in 1925, and in 1926 it was 0·88 per cent. It seemed that now scarlet fever, as it was when described by Sydenham, was hardly worthy of the name of disease. It was interesting, as Dr. O'Brien pointed out, that the disease was more severe in the North. It was mild in France, but the mortality from the disease in this country was not so high as French writers supposed. Even so well-informed a person as Professor Teissier[2] contrasted the malignancy of scarlet fever in this country with its mild character in France. It was especially in South-East Europe that scarlet fever was malignant; in Roumania, Bulgaria and Italy the mortality from it was quite high. The figures, however, must be accepted with some caution. They were to be found in the June and July issues of the *Bulletin de l'Office International d'Hygiène Publique*, especially in the report which was drawn up by Dr. Allan Parson, of the Ministry of Health, and presented by Sir George Buchanan to the International Office.[3] It gave the morbidity and mortality figures from scarlet fever in the various countries of the world. It had been pointed out, however, that in countries with a high case-mortality there was a tendency not to include all the cases; it was rather the severe cases which were included, the milder cases being omitted.

He agreed with what Dr. Goodall had said about not giving the serum in every case of scarlet fever. In studying the literature, he found that several writers urged that this should be done, just as serum was given in practically every case of diphtheria. The concentrated form of the serum was expensive, but apart from this fact there were two objections: (1) (mentioned by Dr. Goodall) that a patient might contract serum disease, which might be more severe than an attack of scarlet fever; (2) that the patient was rendered hypersensitive for some years to come, so that if serum were needed at a future date as a prophylactic against tetanus, or for therapeutic purposes against cerebro-spinal meningitis, the reaction to it was likely to be very violent. For that reason there were only a few cases in which he (Dr. Rolleston) had used the serum. Since March, 1926, out of about 1,000 cases of scarlet fever he had had only sixty-two which he thought required the serum, and those he had divided into classes (*a*), (*b*) and (*c*). In class (*a*) there were twenty-three cases in which there appeared to be immediate and marked benefit from the serum; in class (*b*) were twenty-four cases in which the benefit, though definite, was less marked and rapid; in class (*c*) there were fifteen cases showing no benefit from it, and the deaths numbered five. Serum rash was present in twenty-six cases: in nine there was pyrexia, with some constitutional disturbance, and sometimes there was secondary adenitis and pain in the joints, such as was met with in serum sickness due to other sera.

His dosage, like that of Dr. Goodall, had varied from 10 c.c. to 30 or 40 c.c. Sometimes, but not often, the doses were repeated.

Another point had been suggested to him by Dr. O'Brien's contribution, namely, that of discharging patients earlier if they had had the serum. Was one justified in doing so without a bacteriological examination? He had had no personal experience of the bacteriology of scarlet fever, but in the *American Journal of Hygiene*, and also

[1] *Bull. Off. Internat. d'Hyg. Publ.*, 1926, xviii, 1295.
[2] *Nouveau Traité de Médecine* (Roger, Widal, Teissier), 1922, Fasc. ii, art. "Scarlatine," 1.
[3] *Bull. Off. Internat. d'Hyg. Pub.*, 1926. xviii, 612.

in a German paper, it was stated that *Streptococcus scarlatinæ* persisted for a long time. According to the American observer,[1] it was found to persist at the end of three weeks, and in the case of the German observers [2] it was still persisting at the end of six weeks

Sir FREDERICK ANDREWES, O.B.E., F.R.S.

said that he had had no practical experience in the application of serum treatment to scarlet fever; but in one question he was more interested than in the treatment of scarlet fever itself, namely, that which had been brought forward in Dr. O'Brien's remarks about the overlapping of the effect of scarlatinal antitoxic serum on other streptococcal diseases. It raised a point of fundamental difficulty and importance, namely, whether there was, clinically, such a disease as scarlet fever, i.e., whether it was a sharply-defined entity, or whether it so shaded off into other forms of streptococcal infection that it was difficult to say what was scarlet fever and what was not. During the last thirty-five years, at St. Bartholomew's Hospital, it had been his (the speaker's) duty to issue a report on all cases of infectious disease arising *de novo* in the wards; and in that time he had seen, perhaps, hundreds of cases of doubtful scarlet fever which had originated in hospital; so-called "burn" scarlet fever, cases originating after operations—usually two days afterwards in children—and at the end of that long experience he was left in the gravest doubt as to where scarlet fever left off and where other forms of streptococcal infection began. And in the bacteriological evidence for the streptococcal nature of scarlet fever there was the same difficulty; was it always one particular kind of recognizable streptococcus which caused scarlet fever? In a recent paper Dr. Griffith, of the Ministry of Health Laboratories, had stated his conclusion that at least three different serological types of streptococcus were obtainable from the throats of scarlet fever patients; and the two common in this country were not those which seemed commonest in the United States. But he (the speaker) did not wish to put forward a serological classification of streptococci from the point of view of agglutination as necessarily covering the ground in the diagnosis of scarlet fever. The important thing which had emerged from recent studies in scarlet fever was the recognition that it must be looked upon as pre-eminently a toxic disease. People talked about "toxic scarlet fever" twenty years ago, but that was very different from being able to isolate the toxin of scarlet fever, study it in the laboratory and immunize animals against it. Might there not be a number of serological races of the coccus, all forming one common toxin, as was the case with the bacilli of tetanus and gas gangrene? In the case of the diphtheria bacillus there were a number of races serologically recognizable by agglutination, but forming a common toxin. And might not that toxin be produced by other forms of streptococcus than those obtainable from the scarlet fever throat? The facts which Parish and Okell had published seemed to afford some evidence that such was the case.

It would be interesting to see how far the concentrated scarlet fever antitoxin which Dr. O'Brien was preparing would prove efficacious in conditions allied to scarlet fever. One of the workers on streptococci in his (Sir Frederick's) laboratory contracted what, clinically, was typical acute rheumatic fever, in the summer of 1926. Some of Dr. O'Brien's concentrated anti-scarlet fever serum was administered to her, and, apparently, with strikingly beneficial result. When, later, she had a relapse, a dose of the serum was administered, and in four hours the temperature had returned to normal and the pain had left the joints.

There was much yet to learn about the toxins of scarlet fever and the streptococci generally, and it was along those lines that future advance was likely to be made.

1 E. E. Nicholls, *Amer. Journ. Hyg*, 1927, vii, 84.
2 V. Friedemann and H. Deicher, *Deutsche med. Wochenschr.*, 1926, lii, 2147.

162 Joe—Martin: *Discussion on Serum Treatment of Scarlet Fever*

Dr. Alexander Joe

said he wished to touch on one or two points particularly in regard to the types of the disease.

He agreed that there had not been much opportunity in London of clinically estimating the value of this serum. The disease in London was mild in comparison with what it was in the North—in Edinburgh, for instance. In London he (Dr. Joe) had not been able to show to his students a single case of the septic type. Other complications such as otitis, adenitis, and arthritis seemed to be very similar, but in five months he had not seen a true case of scarlatinal nephritis. For that reason the evidence of Dr. Benson probably carried much weight.

With regard to serum sickness, he had not seen a very severe type of scarlet fever in London, but, until he came to London, he had never realized what serum sickness was. He did not know whether this was due to greater sensitiveness on the part of the children in London, or to the type of serum provided, but in the North there were not so many cases of serum sickness, nor was there such an extreme degree of it when it did occur. This applied, of course, particularly to the treatment of diphtheria. Even so, he (the speaker) did not regard the possibility of serum sickness as a contra-indication for the use of scarlatinal antitoxin in cases requiring it, especially when a concentrated serum was available.

Another interesting point concerned the efficacy of the older serum. He and his colleagues had used it in the very septic cases, but he realized now that the reason why better results had not been obtained was probably that it was given too late. Soon after the Schultz-Charlton work was published, someone had used this old serum, which had produced a definite degree of blanching, thus proving that it contained a specific antibody.

With regard to overlapping, the puerperal serum which Dr. O'Brien had given him had produced in one case a fair amount of blanching in a typical scarlet fever rash. About the time when the anti-scarlatinal serum was first produced he (the speaker) had injected it intracutaneously in the track of a spreading erysipelas, but it did not hold up the spread of the erysipelas. On the other hand, he had isolated a streptococcus from a case of erysipelas which gave the typical reactions of the Dick toxin in a 1 in 1,000 dilution. It produced a high percentage of positive reactions in early scarlatina, and a high percentage of negative reactions in the late days of the disease.

Referring to Sir Frederick Andrewes' remark as to scarlet fever being a clinical entity, he (Dr. Joe) had no doubt that it was such, but he had once heard an ear-and-throat surgeon say that sometimes while the patients on whom he had performed tonsillectomy were contracting scarlet fever, the patients on whom he had performed mastoid operations were beginning to suffer from erysipelas. He (the speaker) did not know whether this was more than a clinical impression, but definite evidence had been produced that evening by Dr. O'Brien of a certain overlapping in the antigenic qualities of these organisms, and it was also possible that the mechanism of infection determined to some extent the clinical picture.

Dr. C. J. Martin, F.R.S.,

asked whether any colleagues had met with an epidemic spread of any other form of streptococcal infection, such as occurred in scarlet fever.

Dr. J. E. McCartney

said that one point raised was as to how long the so-called *Streptococcus scarlatinæ* persisted in the throat. One had visions of people being regarded as carriers, just as in diphtheria, and it was necessary to determine what the infectivity of scarlet fever was before clinicians asked bacteriologists to examine the throats of patients with a view to discharge.

He had always thought there was an overlapping of the group of streptococci. Quite recently one of the maids in a hospital had been suffering from scarlet fever. She had partially recovered from that when she had contracted erysipelas. The temperature became normal, and then pyæmia developed. A hæmolytic streptococcus was isolated from the blood, and the clinician wished to know whether the erysipelas or the scarlatinal streptococcus was responsible for the complication. He (Dr. McCartney) could not answer such a question, because little was known about the inter-relationship of these streptococci. Work must be done on that before the degree of infectivity of these cases of scarlet fever could be determined. Perhaps information would be obtained if a study were made of the organisms in those cases which failed to respond to serum treatment. The scarlet fever serum did not influence certain types of cases, and if those types were studied more and the serological characters observed, another serum could perhaps be made which would exercise a beneficial effect on the cases it was desired to save.

Dr. H. J. Parish

said that further experiments carried out by Dr. Okell and himself appeared to suggest the identity of hæmolytic streptococcal toxins. Rabbits could be killed by the intravenous injection, not only of scarlet fever streptococci, as mentioned by Dr. O'Brien, but also of toxin; large doses of toxin, however—(20 to 40 c.c.)—were required to ensure deaths overnight. If antitoxin were given from four to six hours before injecting the toxin, the rabbits survived. Such experiments had only been possible with scarlet fever toxin, as erysipelas and puerperal fever toxins tended to be much weaker, and there was a limit to the volume of toxin broth one could inject into the rabbit's circulation.

Yesterday he (the speaker) had injected three Dick-positive and two Dick-negative individuals with 1 in 1,000 scarlet fever and 1 in 250 erysipelas and puerperal fever toxins. In each Dick-positive individual the scarlet fever toxin gave the best reaction, the puerperal next, and the erysipelas least. In the Dick-negative persons no reactions occurred with any of the toxins. These experiments on rabbits and in man suggested only quantitative differences between the three toxins.

To parallel the failure of antitoxin to prevent the septic complications of scarlet fever: large amounts of serum did not prevent the development of arthritis in rabbits injected with cultures of hæmolytic streptococci. In practically all the rabbits which were protected against the initial phases of the infection, joint lesions developed at the end of the first week. At first these were slight, but later became very severe; there was usually pus in the joints and neighbouring muscles from which one could recover hæmolytic streptococci.

Dr. Goodall (in reply)

said that he was glad to hear, through Dr. O'Brien, that his (the speaker's) favourable impression of the serum had been confirmed by observers of wider experience. He was doubtful whether the use of the serum would shorten the stay of the patients in hospital to less than four weeks. He had been discharging mild cases at the end

of that period for the last twenty years. If the serum cut short severe cases and prevented complications, a larger number of patients would be discharged after a month.

As regarded the Russian work, Gabritchewsky had used as a prophylactic in certain Russian villages a vaccine consisting of the scarlatinal streptococci, together with the broth in which they had been grown, and, it was stated, with success. The result of this work had not become known in this country because it had been published in Russian. Gabritchewsky died before he had completed his observations, and the war had turned our minds to other matters.

In answer to Sir Frederick Andrewes, there were borderland cases, difficult to diagnose, in every acute infectious disease; but the syndrome, sore throat, punctate erythema and fever, known as scarlet fever, was very definite. That disease gave rise to similar cases and not to others, such as erysipelas. Longstaffe had shown, many years ago, that scarlet fever, erysipelas, puerperal fever and pyæmia were closely connected epidemiologically.

Referring to Dr. Martin's question, there were outbreaks of septic sore throat which were neither due to scarlet fever nor to diphtheria. Some of them were milk-borne epidemics. He would instance the outbreaks in Lincoln, Hackney, and some of the large cities of the United States, for example, Chicago. All were due to streptococcal infections.

He was interested to hear from Dr. Joe, who had now had experience of scarlet fever in both Edinburgh and London, that the disease was more severe in the former city than in the latter. Possibly that was a reason for the more definite results obtained in Edinburgh. The antidiphtheria serum used in the Asylums Board Hospitals was unconcentrated, and more frequent and severe serum rashes were met with than in Edinburgh, where concentrated serum had been used.

Dr. O'Brien (in reply)

said that Dr. Caiger's account of results obtained by the use of serum some years ago made him (the speaker) feel that he had failed to make his point of reconciling what had been done in the past with what was known to-day. The anti-streptococcus serum which had been made for twenty years past apparently protected 50 per cent. of rabbits in the Parish-Okell test. It was practically certain that the serum Moser used contained antitoxin. None of that was available now, but one could test the material made a few years ago. The Dick serum had been first made in April, 1924, but we had to-day a serum made before that date, by the old methods of Moser and others, and this serum clearly protected rabbits against scarlet fever streptococci. The case was parallel to the time, in 1894, when diphtheria serum was first introduced. In the hands of many people it was a failure, but in some hospitals it produced dramatic effects. The explanation was that the serum available in this country was a very low-grade serum. It cured some cases, but it did not contain enough antitoxin to be generally convincing.

With regard to the bacteriology of this disease, Gordon, of Chicago, had recently reported that of 100 scarlet fever cases, sixty-two among the control group who had not been treated with serum, and thirty-one who had been so treated, had the hæmolytic streptococcus of scarlet fever in the throat on the twenty-eighth day. Scarlet fever patients had apparently been discharged from hospitals with hæmolytic streptococci in their throats for years past, but they were not acutely infectious, or "return" cases would more often occur.

With regard to Dr. Martin's question, military records showed that there had been epidemics of sore throat without a rash in groups of soldiers confined to

barracks, and that these had spread over large groups of men. One such had occurred in the South African war.

Reverting to the old work on this subject, Dr. Gabritchewsky's death, in 1905, was to be regretted. It was most refreshing to read his work and to note the logical way in which he passed from one step to another. He had produced a highly successful anti-scarlet fever vaccine.

With regard to the cases mentioned by Dr. Parish, the septic element of scarlet fever was the important one. The deeper one went into it the more difficult it became, and the more clearly it was seen that fascinating problems lay ahead. Possibly there was an overlapping in, or even identity of, the antigens of all the hæmolytic streptococci, but the baffling thing to-day was the occurrence of later septic phenomena. In the rabbits experimented upon, the first attack of the streptococcus was neutralized by the serum, and the protected rabbits were alive and well after the control rabbits were dead, but later joint lesions had developed. This curious paradox was first recorded, in connexion with pneumonia, by Mair and Gaskell. One could immunize a rabbit with dead pneumococci at first, then with the living pneumococci, and the rabbit's serum would protect a mouse against many lethal doses of pneumococcus; endocarditis and other pneumococcal lesions developed in the rabbit itself. Until a means of preventing this late arthritis in the rabbits was discovered, it would not be possible to avoid these serious complications. Dochez had sent to him (the speaker) some of his antibacterial serum, and he had tried that and other sera supposed to be antibacterial and containing a large amount of agglutinin, but those sera had not saved the rabbits from this late arthritis. The worker who could solve the problem of preventing these complications would earn a great debt of gratitude.